University of Toronto

# MEDICAL JOURNAL

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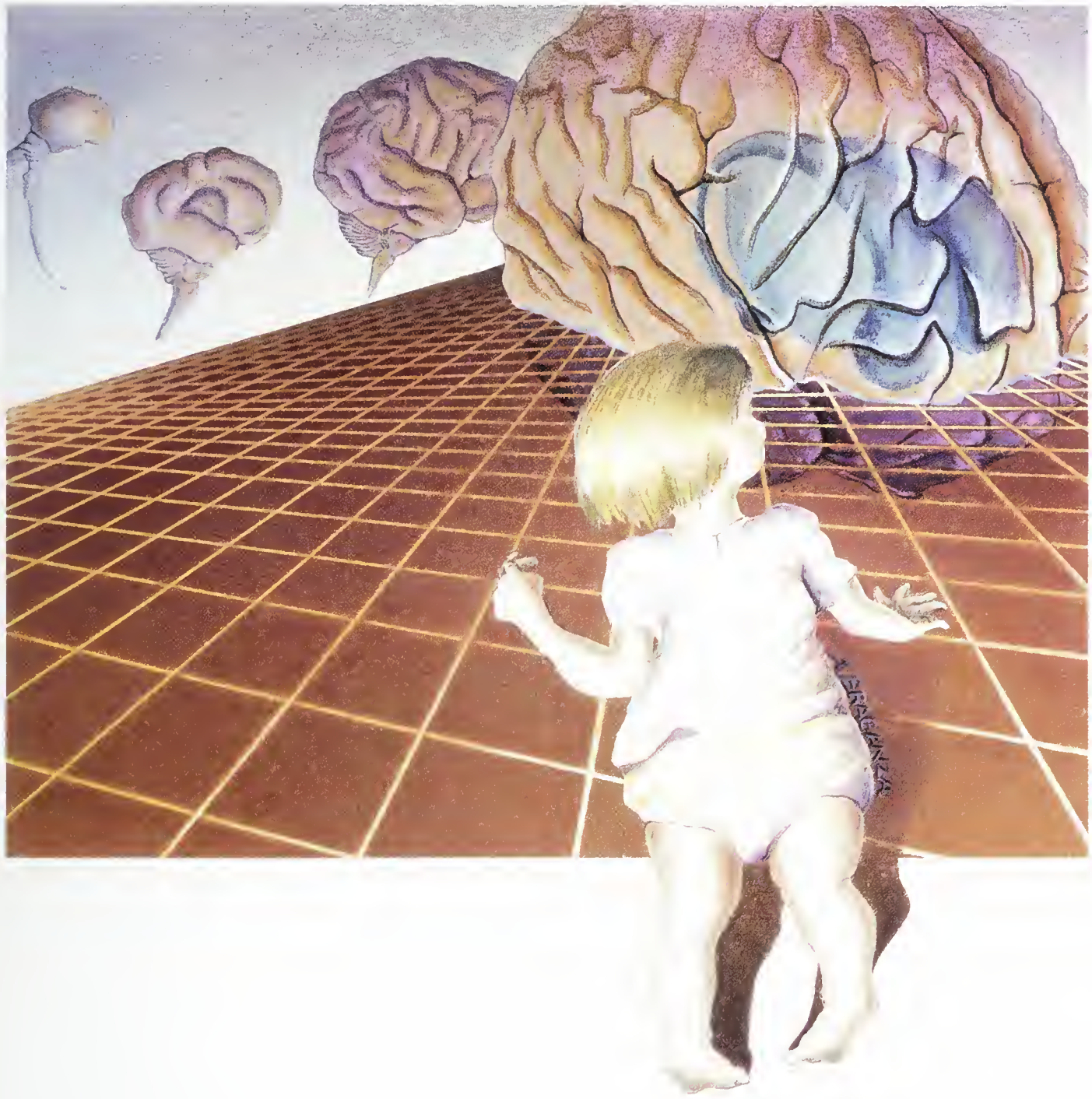

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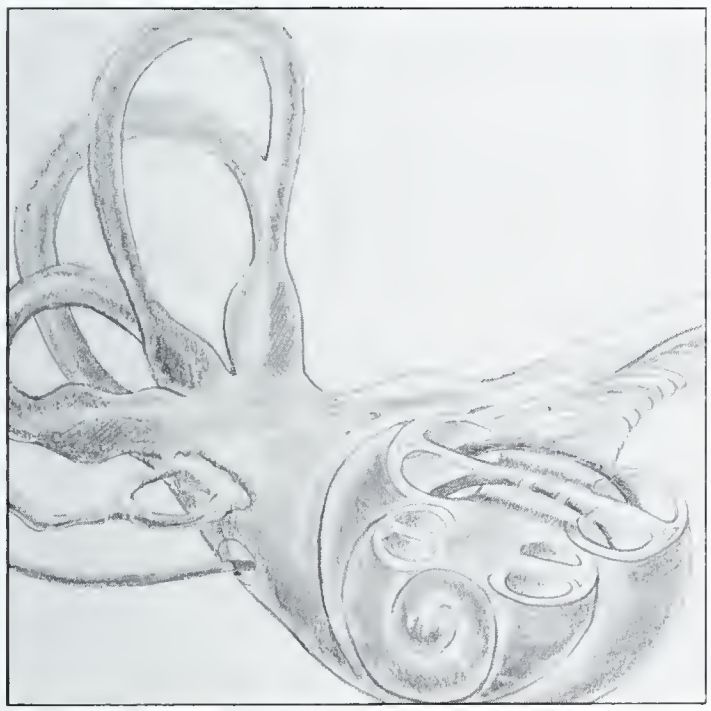

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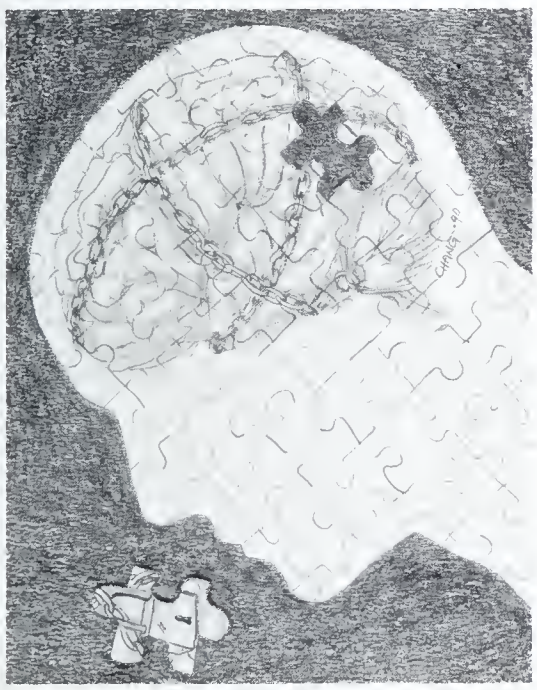

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# Editorial

*Filiberto Altomare*
*Mitesh Mehta*

We feel that the first edition of the *University of Toronto Medical Journal* was an outstanding success. We received many excellent articles and it was difficult for our editorial board to choose the best ones from among them. However, this does not mean that we have stopped soliciting articles. On the contrary, we encourage you to submit articles as this *Journal* provides a necessary forum for student publications.

The *Journal* would like to announce the 1st annual Biomedical Ethics Competition. Papers between 1,000 and 2,000 words on biomedical ethics are solicited. Papers should be sent to:

Dr. John Sen
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A panel comprised of faculty and *Journal* staff will select the best paper in the field of biomedical ethics. This paper will then be published in the first issue of the 68th edition of the *Medical Journal*. The cash prize that accompanies this is $500 and the deadline for submission is April 30. This contest is open to University of Toronto Medical students only.

This issue of the *Journal* surpasses any produced so far. In this and hopefully all upcoming issues, the abstracts are printed in both English and French. We feel this adds a more Canadian flavour to our *Journal* and will increase its readership. We would like to thank Monique Lafrenière 9T1 for her help in the translations.

In this issue of the *Journal* we have excellent articles on gait analysis in cerebral palsy, somatoform disorders, cochlear implants, and the effect of aerosolized pentamidine in HIV-seropositive patients. In addition we have our regular features, Quick Dx, News Updates, Rounds, Book Reviews and Computers in Medicine.

Once again we would like to thank our staff for all their hard work in preparing this issue. We would again like to encourage those among you who may be interested in submitting a paper to do so. This student run publication is also student written and we would like it to remain so. We hope you enjoy this issue.


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# Cochlear Implant Design and Indications

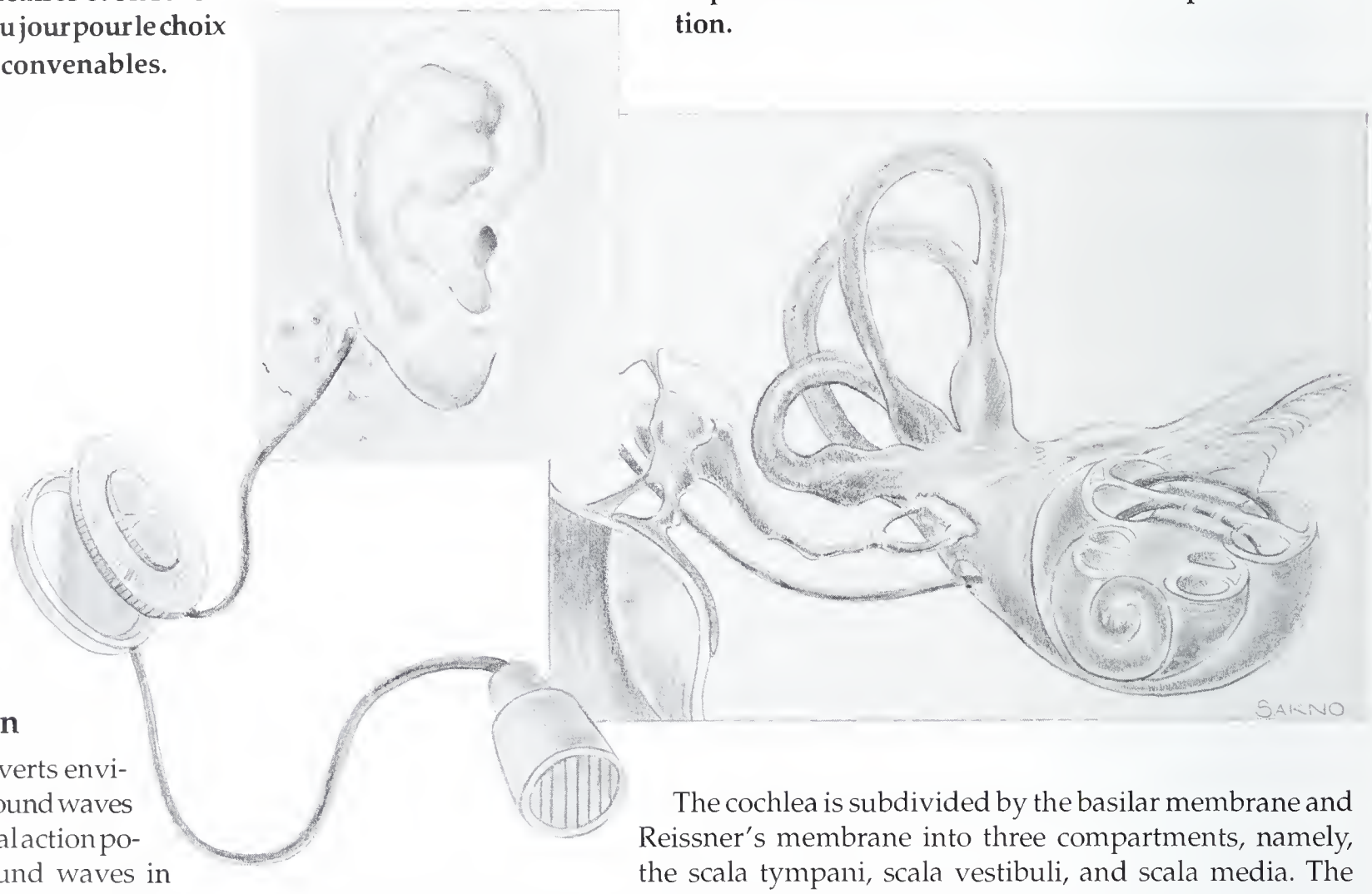
*Alden J. Pearl 9T1*


## Abstrait

L'implant cochléaire est une prothèse utilisée pour le traitement de la surdité sensorineural profonde. Cet appareil détourne le système transductif de l'oreille et stimule le nerf auditif directement. Certains patients récipiendaires de cette technologie ont fait l'expérience d'une ouïe améliorée. On présente ici les desseins actuels des implants cochléaires et on revoit les critères du jour pour le choix de patients convenables.

## Abstract

The cochlear implant is a prosthesis used in the treatment of profound sensorineural deafness. This device bypasses the ear's transduction system to directly stimulate the auditory nerve. Although limited, this technology has been demonstrated to improve hearing in selected patients. This paper reviews the present designs of cochlear implants and outlines current criteria for patient selection.


## Hearing & Sound Perception

The ear converts environmental sound waves into neuronal action potentials. Sound waves in the external ear are transmitted to the tympanic membrane where subsequent vibrations initiate ossicular motion. Ossicular movements set up waves in the fluid of the inner ear. The fluid waves act on the cochlea's organ of Corti to generate action potentials in the auditory nerve fibres.

The inner ear, or labyrinth, is composed of a system of chambers filled with fluid. The cochlear portion of the labyrinth, as opposed to the vestibular portion, is responsible for the transmission of sound to higher neuronal levels. The cochlea (snail) is a 35 mm channel coiled into 2-1/2 turns around a central bony axis. The turns are designated as basal, middle and apical. The basal turn is widest, closest to the stapes, responsible for the perception of high frequencies, and bulges into the middle ear space as the promontory.

The cochlea is subdivided by the basilar membrane and Reissner's membrane into three compartments, namely, the scala tympani, scala vestibuli, and scala media. The sensory units of hearing, specialized hair cells, are embedded within the basilar membrane of the cochlea's organ of Corti. Consequently, motion of the basilar membrane from fluid waves stimulates the sensory cells to generate nerve impulses in the cochlear nerve.

The analysis of sound begins peripherally in the cochlea, via frequency selection along the basilar membrane and is termed place or resonance theory. The basilar membrane is composed of fibres of variable length, shortest at the basal end and longest at the apical end. Each fibre will vibrate maximally at a given frequency. Thus, the cochlea's frequency selection is based on the natural resonance of a given fibre, which stimulates a particular set of hair cells. In addition, the "travelling wave" theory states that as the sound progresses through the cochlea, the whole basilar

membrane vibrates, but the maximun amplitude of resonance occurs at a specific point on the basilar membrane constant for a given frequency. As the complex "noise" wave travels, its component waves stimulate frequency specific areas of the basilar membrane and hair cells of the organ of Corti. The final interpretation of different sound frequencies and their reconstitution into a complex noise occurs in higher brain centres.

Clinical deafness due to impaired sound transmission in the external or middle ear is termed conduction deafness. Sensorineural deafness occurs when there is damage to the cochlear hair cells or central neural pathways. It is sensorineural deafness due to cochlear damage that is addressed with cochlear implant technology.

## History

The concept that the auditory system can be directly stimulated by an electric current is an old one. In 1790, Volta inserted metal rods into each of his own ears and connected them to a circuit. Upon closing the circuit he felt the sensation of a head blow, which was followed by the sound of a boiling viscous liquid.[1]

The first attempt to produce hearing by electrical stimulation of the auditory apparatus was in 1957 by Djourno and Eyries. Although their results were optimistic, their work did not receive much attention.[1] Fortunately, new developments in microsurgical techniques and increased knowledge of biocompatible materials have stimulated others to pursue the creation of a true cochlear implant. Subsequent work in the sixties progressed to the experimental implantation of a handful of devices. Whereas there were fewer than 100 implants by 1977, to date there have been over 3000 implants performed worldwide.[2]

## Cochlear Implant Systems

The growing interest in cochlear implants has resulted in the development of a wide variety of devices. Despite the diversity of current implants, all operate on the same principle. Although the inner ear's hair cells may be damaged or diminished in sensorineural deafness, some cochlear neurons may remain intact. It is these viable neurons that can be directly stimulated by an externally produced current. The subsequent nerve impulses travel along the auditory pathways to the brain which interprets these as sound.

All cochlear implant systems also share the same basic components (Fig. 1). An external microphone receives sound and delivers it to a speech processor. The function of the cochlear hair cells is reproduced by the processor which transforms acoustic energy into an electric signal. This information is then relayed to the implanted component to excite the cochlear neurons of the auditory nerve. In most implant systems, a transmitter is placed on the mastoid to relay the processor signal across the skin to the implanted receiver. The transfer of information may be percutaneous (directly through the skin via wires) or, more typically, transcutaneous where an implanted receiver is inductively coupled with an external transmitter.

The stimulus is delivered to the cochlear neurons by either a single electrode or multiple electrodes. With a single-channel system, a single electrode delivers one type of signal to one area of the cochlea. In multichannel designs, differently processed information is conveyed to different electrodes through separate pathways. These multichannel systems attempt to simulate the tonotopic arrangements within the cochlea.[2,4]

In extracochlear systems, electrodes are placed external to the cochlea either on the round window membrane or on the promontory. Such implants are usually single channel in function. Although extracochlear implantation is less invasive, it requires greater amounts of current to generate its signal. This increased current can produce potential side effects including otalgia and vertigo.[4]

Intracochlear stimulation refers to those systems which are placed in the scala tympani and make contact with cochlear dendrites.[4,7] In contrast to extracochlear implants, intracochlear implants are technically more difficult to insert, induce further degeneration and promote new bone formation.[8,9] Since intracochlear electrodes may be either single- or multichannel, the greatest variation in implant design is possible with intracochlear, multichannel systems.[2]

Both an active and a ground electrode are required to complete the electrical circuit. Consequently, implant electrodes are designed in either a monopolar or a bipolar configuration. In monopolar stimulation the active electrode is near the cochlea while the ground is outside the cochlea. By separating the electrodes, a relatively broad area of the cochlear nerve may be stimulated.[4] With bipolar configurations, the electrodes are placed adjacent to one another, often within the cochlea, in an attempt to limit the spread of electrical current to one region of the cochlea. Theoretically, such a design limits the interaction of multiple signals and eliminates any uncomfortable additive effects on loudness perception.[4] However, due to the sur-

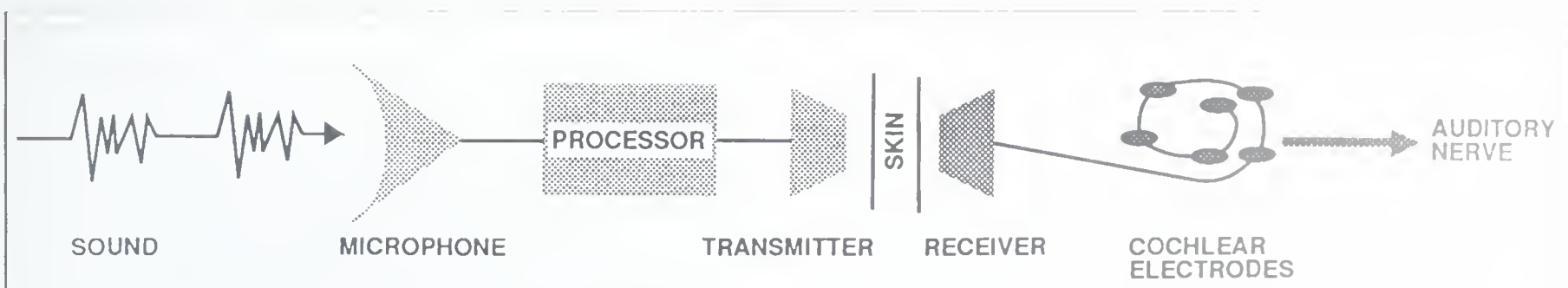



**Figure 1:** Schematic representation of a cochlear implant system.

face requirements of electrodes and the limited cochlear area, bipolar electrodes account for up to 10-15% of the electrodes in multichannel implants.[4]

## Speech Coding Strategies

The function of the inner ear is to extract acoustical information from sound energy and transform it into electrical energy. Neural channels deliver this processed information to the brain for interpretation. The cochlear implant attempts to simulate this complex encoding with only a fraction of the neural channels using nonphysiologic electrical stimuli.

Whereas the phonetic features of speech are well known, the mechanisms by which the ear processes speech is largely speculative. Consequently, a variety of speech coding strategies can be found among the available cochlear prostheses: analog transformation, vocoder processing or feature extraction algorithms.

The analog model attempts to present as much of the acoustic signal to the cochlear nerve as possible by deriving a signal "more-or-less" directly from the analog waveform of speech.[3,4] It is hoped that by "squeezing" as much information as possible into a single channel, the patient will learn how to make sense of it even if the sensation is very unnatural. Unfortunately, by squeezing all the acoustic cues into a single channel, it seems likely that the user will only be able to extract a very limited proportion of the available information.[5]

Practically, the understanding of speech occurs in the presence of background noise. Normally, the cochlea plays a major role in separating the sounds of speech from those of noise thereby functioning as a frequency selectivity mechanism. In the analog model, signals can only be discriminated on the basis of their temporal pattern, and background noise is processed along with the desired speech.[4,5] The resultant signal does not separate speech from noise such that, implants using this scheme are of limited use in noisy environments.

Vocoder processing provides acoustic information by separating the complex acoustic signal into separate bands. The output from each band filter determines the signal that is delivered to electrodes positioned tonotopically along the cochlea. The objective is to approximate the normal filtering ability of the cochlea's basilar membrane.

In principle, vocoder processing schemes are capable of delivering more speech information than those using analog transformation. However, the assumption that there are some neurons surviving at each electrode position is often not true as there may be only patches of intact cells.[5] Secondly, there are anatomic constraints with the placement of multiple electrodes along the cochlea. Finally, the temporal patterns of neural firing crucial for the coding of pitch are created by electrical stimulation, and are different from those evoked in the normal ear by the acoustic signal.[5] It should be appreciated that the temporal mismatch is not an exclusive problem of the vocoder processing but common to all speech coding algorithms.

**Table I Indications for Cochlear Implantation[4,10]**

| | |
|---|---|
| 1 | Bilateral profound to total sensorineural hearing loss |
| 2 | Unable to benefit from conventional hearing aids |
| 3 | No radiographic contraindications |
| 4 | Patient must be in good physical and mental health |
| 5 | Patient must have the motivation and patience to comply with a lengthy rehabilitation |

Feature extraction approaches are based on the theory that certain aspects of the acoustic signal are sufficient for speech identification. Consequently, these processors use filters to extract certain frequency-dependent features of speech and present these in isolation.[3,6] Separating out only those features necessary for word understanding may make the identification of acoustic information easier than if the entire signal is presented.[3] A further advantage of this scheme is that the electric "sounds" can be successfully delivered even in background noise with little interference.[5]

With feature extraction, the processor supplies limited yet selected information complementary to that visible on the speaker's face such as positioning and movements of the tongue, lips and jaws. However, there is no visible correlate of voice intonation and voicing, which is a product of vocal cord movement. Implants include pitch extractors to overcome such apparent limitations.[5] These devices evoke single pulses for each period of vocal cord vibration, thereby approximating voicing and the related feature of voice intonation.

It must be appreciated that there are a number of implants available utilizing the various strategies outlined. Each system offers advantages as well as drawbacks. It follows that of the available implants no one has been shown to be consistently superior to the others.[4]

## Patient Selection

Most implant groups use similar criteria for patient selection (Table I). Such criteria include the extent of one's hearing loss, poor efficacy with conventional hearing aids, and anatomy which is not restrictive to the required surgical procedure.

The primary means of determining implant suitability is an audiologic assessment. In general, implantation is limited to the bilaterally deaf with the average hearing threshold for pure tone stimuli (500 Hz, 1000 Hz and 2000 Hz) of 90 dB hearing or poorer[4,10,11] where normal hearing thresholds range between 0-20 db. Patient selection not only depends on the pure tone average threshold but also on the inability to benefit from conventional hearing aids. The subject would have to receive significantly less word recognition with the use of conventional devices than would be expected from a cochlear implant.[10,11]

**Table II Psychological Criteria for Cochlear Implantation in Children**[10,12]

1. No evidence of severe organic brain damage
2. No evidence of mental retardation
3. No evidence of psychosis
4. No evidence of behavioral personality traits that would make completion of the rehabilitation unlikely
5. Realistic expectations about the implant on the part of the family
6. Well ordered family environment for post implantation rehabilitation and support

The medical history should address both the patient's general health and determine the etiology and onset of deafness. It is generally appreciated that the onset of deafness is the major factor influencing subsequent performance in both adult and child implant candidates. The onset of profound sensorineural deafness has been defined as follows:[10,11]

**1** Prelingual deafness – congenitally present (under 1 yr) with no acquisition of language skills and no established auditory memory.

**2** Perilingual deafness – sounds heard but hearing lost prior to the development of language (1-5 yr); may have auditory memory.

**3** Postlingual deafness – hearing loss after developing verbal language skills.

The majority of programs limit their patients to postlingually deafened individuals. Some groups select perilingually deafened subjects who post-operatively show a similar gain of auditory information as compared to the postlingually deafened.[10,12] However, a significant number of prelingual deaf patients cannot use the extra auditory information provided by the cochlear implant as effectively. Careful counselling and post-operative rehabilitation is required because many have stopped using their implants.[11,12]

It had been suspected that the etiology of acquired deafness would have an impact on performance. However, it has been reported that there is no significant difference based on etiology as a predictor of subsequent performance.[11] Nevertheless there are some aspects of etiology which may be useful.

The integrity of the auditory nerve is crucial for implant candidates and varies with the etiology of deafness. Patients with a history of meningitis or labyrinthitis have limited cell survival and should be forewarned that there is a possibility that performance may be below average.[11] These patients as well as those with a prior temporal bone fracture should undergo electrical stimulation to determine the nerve's integrity.[10,11] A positive response which is generally indicative of suitability to implantation, is a perception of sound by the inner ear when either the round window or the promontory is stimulated.[11] Responses with stimulation of the promontory have been inconsistent – both patients with negative or positive tests show auditory responses to intracochlear implants.[10,11] Although preoperative electrical stimulation is useful in assessing the integrity of the auditory tract, it is not predictive of future performance of implant candidates.

Prior to implantation, it is important to examine the candidate and identify any external or middle ear disease. Tympanic membrane perforations and otitis media must be treated preoperatively. Also the diagnosis of otosclerosis or a history of previous ear surgery should alert the surgeon to the possibility of distorted anatomy.

In all candidates, computed tomography of the temporal bone is performed to identify ossification or obliteration of the scala tympani, congenital malformation of the inner ear and surgical landmarks.[13] Complete agenesis of the cochlea or an abnormal acoustic nerve is a contraindication to the placement of a cochlear implant.[10,11,13] Bony obstruction of the scala tympani is not necessarily a contraindication as cochlea with partial obstruction to near total obstruction have been implanted.[14,15] Furthermore, there has been at least a single case report suggesting that labyrinthectomy is not a contraindication to auditory rehabilitation using a cochlear implant.[16]

Higher level cognitive skills are suspected to be related to implant success.[11] In adults and especially in children, certain psychological criteria must be met (Table II). Generally, paediatric candidates must be neurologically intact and be part of a parent-child interaction where the family will be able to follow through with the child's post-implant rehabilitation. Nevertheless, implantation in paediatric patients is fiercely debated and is performed in a very limited number of centres.

## Other Issues

While some surgeons implant the poorer hearing ear others implant the most recently deafened ear – usually the better hearing one.[11] This ear is the one that responds to the greatest number of frequencies, obtains a better response on electrical stimulation and is subjectively perceived to sound better with or without a hearing aid. In addition, the ear without prior surgery, with the least amount of bony obstruction and noncongenital hearing loss is deemed a better candidate. The ear with the greatest residual neural cells and requiring the least technically difficult surgical procedure is chosen for implantation.

The cochlear implant design and approach is vastly debated.[4] Ideally, there should be an attempt to match device features with candidate parameters such as residual nerve function and cognitive processing characteristics. Current data is insufficient to draw sweeping conclusions because predictors of performance are not agreed upon.[4] Despite the implants' theoretical basis there are many aspects of this technology which are neither fully understood nor appreciated.[7]

Extracochlear multichannel implants may provide more information than intracochlear single channel systems in the presence of residual hair cells.[4,7] All implant designs enhance lip reading and awareness of environmental cues

which is of great benefit, especially regarding personal safety.[4] Patients using the multichannel implants recognize some speech without visual cues and consistently perform at higher levels of audiologic function.[4,11]

Nevertheless, performance varies among patients within implant groups. In selected subjects, multichannel implants enable effective communication by sound only – an explanation is unknown but may be related to a higher residual auditory nerve function and superior cognitive skills.[4]

## Summary

It must be appreciated that cochlear implant technology is still in its developmental infancy. There are a number of devices which, with proper training, can be useful in rehabilitating postlingual, profoundly sensorineural deafened individuals. Issues on future development of cochlear implants include optimization of design and speech coding, development of preoperative predictors of performance and implementation of this technology in the prelingually deaf and in children.

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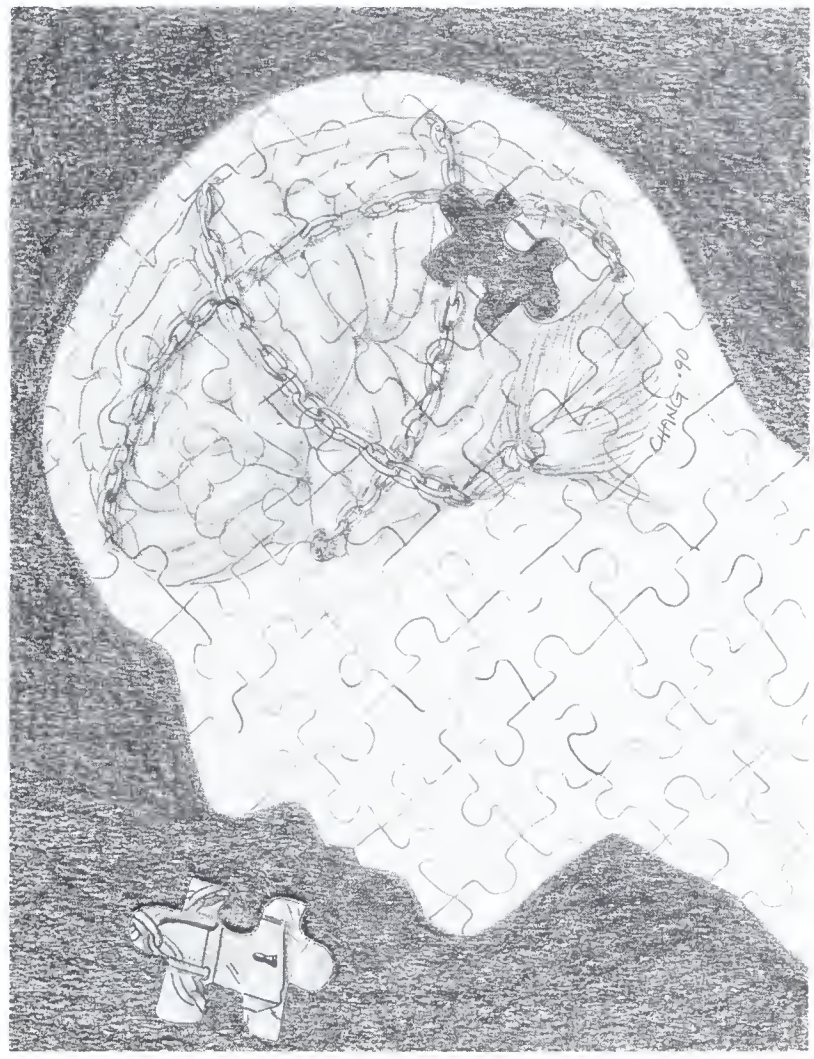
# Somatoform Disorders and Other Psychiatric Disorders Associated with Somatization: A Review with Illustrative Case Studies

*Michael I. Singer B.A. 9T1*


## Abstrait

**Plusieurs patients se plaignent de problèmes fonctionnels somatiques. Cette revue examine les principaux troubles somatiques et psychiatriques qui font partie du diagnostic différentiel des phénomènes de somatisation. Trois cas seront décrits afin d'illustrer les éléments caractéristiques de ces entités cliniques. On a conclu que certains aspects psychiatriques, somatiques et medicaux peuvent coexister à l'intérieur d'un seul patient.**



## Abstract

**Many patients present with functional somatic complaints. This article reviews the major somatoform and psychiatric disorders which may form part of the differential diagnosis in a patient presenting with somatization phenomena. Three cases are described to illustrate the main features of these clinical entities. It is concluded that aspects of several psychiatric, somatoform and medical disorders may coexist within the same patient.**


## Introduction

Somatization involves the expression of psychic distress in physical terms. Although it is often transient and not necessarily pathological, somatization becomes a clinical and socioeconomic problem when an individual becomes incapacitated or preoccupied with his bodily experiences, attributes them to medical illness and seeks diagnosis and treatment.[1] It has been estimated that at least 30% of primary care patients consult physicians for functional somatic complaints.[2]

Somatization can be a major feature of a number of psychiatric syndromes. This article will discuss the somatoform and psychiatric disorders which may form part of the differential diagnosis for a patient presenting with somatization phenomena. Three cases will be presented to illustrate the salient features of these interesting clinical entities.

## Somatized Depression

The presentation of major depression with somatic symptoms may occur in 25-50% of severely depressed patients.[2,3] Depressive equivalents can mimic virtually any organic disease.[2] A somatized or "masked" depression should be suspected when:

1 The patient's complaints mimic but do not follow the typical picture of a physical disorder and a diagnostic workup fails to confirm an organic disease;

2 The symptoms do not respond to established treatments for the suspected illness;

3 The patient's symptoms shift – as soon as one symptom is gone, another replaces it.[3]

If depression is suspected one should look for associated symptoms such as inability to experience usual pleasures, insomnia, changes in appetite and body weight, diurnal variation of symptoms (worse in the morning), lack of energy, poor concentration and memory, pessimism, tendency to self devaluation, a sense of guilt and failure, suicidal thoughts, evidence of a recent loss or recent onset of a major illness, and a family history of affective disorders.[1,3]

## Somatized Anxiety

Somatization and anxiety disorders are closely linked. Anxiety disorders feature excessive and unwarranted fear or apprehension and as a rule are accompanied by physical symptoms such as dyspnea, palpitations, chest pain, sweating, faintness, dizziness, paresthesia, tremor and fatigue.[1] Patients with anxiety disorders may differentially attend to their somatic symptoms instead of their anxious mood.

This could result in the fear or conviction that one has a disease.

## Hypochondriasis

Hypochondriasis is an unrealistic preoccupation with the fear or belief that one has a serious disease despite negative findings, a benign course over time and appropriate medical reassurance. Hypochondriacs are reported to constitute between 3-14% of all patients seen in general medical practice.[4,5] These patients do not understand the benign nature of their functional somatic symptoms and interpret them as evidence for disease. Hypochondriacal concerns may begin with over-awareness of bodily sensations and can progress to mild apprehension, phobic concern, overvalued ideas and delusions.[3] Hypochondriasis can be an autonomous disorder or secondary to other psychiatric disorders (for example, depression or anxiety disorder). Patients with primary hypochondriasis are also anxious or depressed but the preoccupation with the fear of disease or false belief in having a disease persists and is the most important feature of their psychopathology.[6] They differ from schizophrenics in that their disease conviction is not of delusional intensity (i.e. they can entertain the possibility, if only briefly, that their fear or belief of having a serious disease is unfounded, their symptoms fluctuate more over time and they are not out of keeping with societal norms).[4]

## Body Dysmorphic Disorder

Patients with this disorder gradually become preoccupied with the belief that they are physically deformed or defective in some way. They are frequently concerned about their face, nose, breasts or genitals and they may visit dermatologists and plastic surgeons repeatedly to correct the imagined defect. Their appearance is objectively normal or if they do have a slight physical anomaly their concern is considered excessive. In order to make the diagnosis, it should be determined that the false belief is neither of delusional intensity, nor due to anorexia nervosa or transsexualism.[4]

## Somatization Disorder

Somatization disorder is a relatively new diagnosis which subsumes the old diagnosis of Briquet's syndrome or hysteria.[5] It is reported to occur in 1-2% of all females.[4,5] Typically, these patients have dramatic and complex histories of multiple symptoms, hospitalizations and operations, and often there is a past history of other psychiatric and drug abuse problems. They are prone to repetitive conversion symptoms (a loss or change in bodily functioning that results from psychological conflict or need), and may even acquire a "gridiron abdomen" from numerous procedures. Their childhood was typically chaotic and included marital instability, physical and/or sexual abuse and parental substance abuse.[5] Diagnostic clues of somatization disorder include a family history of alcoholism, an "hysterical" presentation that is dramatic but precise and a past medical history that is vague, disorganized, and full of inconsistencies. These patients apparently have a need to react to what they believe the clinician wants from them. They are, therefore, very suggestible and tend to create a sense of urgency in their physicians which leads them to obtain unnecessary and sometimes dangerous and invasive procedures.[5]

## Malingering

Malingering occurs in normal people and in persons with psychiatric illness but it is most frequently found in sociopaths.[5] Malingering implies fraudulent illness behaviour whereby the patient consciously fakes disease or disability in order to receive some tangible secondary benefit such as relief from military service or financial compensation.

## Factitious Disease

The primary feature of factitious disease is the conscious and deliberate causation or simulation of disease states due to unconscious motivations.[5] It is distinguished from malingering in that there is no ulterior motive or material benefit other than assuming the sick role. Typical characteristics of patients with factitious disease include being young, unmarried and female and having a history of work in the health care professions. The diagnosis is further suspected when the patient reports a lack of close warm relationships.[5]

Factitious disease is a heterogeneous disorder. Some patients demonstrate stability of family and work life while others may be borderline or psychotic.[7] Their production of a disease state may have different psychodynamic explanations. It may be a desperate attempt to create some sort of identity; being a sick person is better than being nothing at all.[5] Factitious disease may also be a more acceptable way for the patient to receive care from symbolic parental figures (doctors and nurses). For other patients inviting painful diagnostic and surgical procedures may serve as a masochistic defense mechanism to diminish unconscious guilt. As well, by frustrating physicians patients may utilize this disorder to gain mastery over parental figures.[7] Others add that patients with factitious disease are motivated by hypochondriacal preoccupations. By producing artificial symptoms these patients can convince their physician to continue to search for the real illness and to take care of them.[8]

## Conversion Symptoms and Disorder

Conversion disorder is a somatoform disorder involving the change or loss of physical functioning that suggests an organic disease but that is apparently an expression of psychic conflict or need.[4] Conversion symptoms are common with incidences ranging from 5-30% in hospitalized medical or psychiatric patients.[9] These symptoms are said to substitute for and reduce anxiety, being symbolic of a conflict involving aggression or sexuality, and they often provide secondary gains for the patient.[10] Some clinicians suggest that the presentation of these symptoms consti-

tutes a form of interpersonal communication or manipulation[11] while others argue that they are merely conditioned behaviours.[10] Conversion symptoms can also occur as part of a medical disorder as they can complicate a physical illness and can prolong recovery. The diagnosis of conversion disorder is established by excluding organic disease, determining that the symptoms are not under voluntary control, and demonstrating contributing psychological factors.

## Somatoform (Psychogenic) Pain Disorder

In somatoform pain disorder, pain is the primary complaint. It is related to environmental stress and is either inconsistent with known neuroanatomic distribution and pathology or is grossly in excess of what would be expected from whatever physical findings are present.[4] These patients typically have difficulty experiencing and expressing feelings. Emotionally stressful events are apparently immediately translated into somatic symptoms.[10]

## Case 1

A 19 year old male from rural Ontario presented with right hand and wrist pain, decreased mobility and decreased sensation subsequent to a fist fight 1-1/2 years earlier. He had been taunted by a fellow student at his high school for the previous five years. Finally, the patient hit him and apparently broke the student's nose and rib and caused his eye to bleed. The patient was left with a painful swollen hand and wrist after also punching a cement floor. He was examined at a local hospital and was eventually given the diagnosis of Reflex Sympathetic Dystrophy (RSD), a deafferentation syndrome that occurs following injury to bone and soft tissue. Two nerve blocks were performed to relieve pain but the patient said this did not help. He experienced substantial disability: not being able to write, quitting his job, graduating a half year behind his peers and postponing university for a year. He was finally sent to Toronto General Hospital (T.G.H.) where doctors investigated his complaints but found no physical reason to explain the degree of disability he was experiencing. His numbness and pain did not follow normal dermatomal distribution. A sodium pentathol test showed almost full mobility of the affected hand. Stellate ganglion blocks were done and the patient experienced only one day of pain relief (most patients with RSD experience relief for 72 hours to 1 week). The results of a bone scan were equivocal, x-rays were almost totally normal except for some mild osteoporosis and the patient exhibited no atrophy, sweatiness or dryness of skin with nail changes. These findings made a diagnosis of RSD doubtful.[12,13]

The patient said he did not feel depressed or anxious, there was no loss of appetite or energy nor any sleep disturbances. There was also no history of psychotic symptoms, no alcohol or drug addiction, no self harm, suicidal or homicidal tendencies. Nevertheless, the patient's reaction to the fight seemed curious. He regretted having "let go" and hurting someone and actually allowed the person he hit to punch him in the face in return. Similarly, two years previously he was instructed to break an opposing player's leg during a football game, did so and later quit playing because he felt so badly about what he had done. The patient admitted to a reputation for being very aggressive on the playing field.

On mental status exam, the patient was found to be polite, cooperative and somewhat shy. There was no evidence for a mood disorder, no psychomotor retardation or agitation. He was alert, oriented and had no psychotic symptoms but he was very preoccupied with his hand and had great difficulty expressing affect.

**Table I Somatoform Disorders & Other Psychiatric Disorders Associated with Somatization**

- **Mood Disorder (somatized depression):** atypical presentation, poor or no response to treatment, shifting pattern, associated typical depressive symptoms
- **Anxiety Disorder (Somatized Anxiety):** excessive fear and apprehension. As a rule, anxiety disorders are accompanied by physical symptoms
- **Psychotic Condition (Somatic Delusions):** bizarre fixed false beliefs inconsistent with cultural norms
- **Hypochondriasis:** unrealistic fear or belief of having serious medical illness despite appropriate medical reassurance
- **Body Dysmorphic Disorder:** imagined physical defect or deformity
- **Malingering:** conscious and fraudulent simulation of disease, often found in sociopaths
- **Factitious Disease:** conscious simulation of disease but due to unconscious motivations
- **Conversion Disorder:** loss or change in bodily functioning resulting from unconscious psychological conflict or need
- **Somatoform Pain Disorder:** preoccupation with severe and continuous pain that has no adequate medical explanation and is presumed to be due to psychological stress

### Discussion

The above patient's problem was both psychological and physical. The patient's complaints of pain, decreased mobility and numbness had no organic explanation 1-1/2 years after his injury yet, at the time of the initial trauma, they were probably due to the physical effects of the insult. The early injuries and symptoms were apparently protracted by the patient's anxiety about his aggressive act and his need to be rendered powerless to commit future aggressive acts. His physical injuries likely facilitated the emergence of conversion symptoms in the same location, demonstrating the process of "somatic compliance".[10]

There was some secondary gain for the patient to remain disabled. He was excused of his responsibilities at school and at work and was catered to by his family and friends. As well, there was previously some family conflict surrounding his travelling away from home to attend university – his mother wanted him to go to a local college, the patient was accepted to study at a university in Toronto – and by assuming the sick role, he was able to delay his decision and thus avoid family turmoil and possible separation anxiety.

The patient's symptoms and situation were found to be consistent with a DSM-III-R diagnosis for conversion disorder:[14]

**A** The predominant disturbance was a loss of and alteration in physical functioning suggesting a physical disorder i.e. Reflex Sympathetic Dystrophy.

**B** Psychological factors were judged to be etiologically involved in the symptom.

**C** The symptom was not under voluntary control.

**D** The symptom was not a culturally sanctioned response pattern and could not, after appropriate investigation, be explained by a known physical disorder.

**E** The symptom was not limited to pain or to a disturbance in sexual functioning.

Management of this patient consisted of avoiding argument as to whether his symptoms were entirely physical or mental. It was suggested that psychological factors might be fuelling his pain and disability and that he might benefit from some individual counselling.

By the end of his hospital stay, the patient was showing a willingness to cope with his disability and to get on with his life and he even began to think of starting university in the fall.

## Case 2

A 65 year old retired engineer presented to T.G.H. with an eight month history of retrosternal chest pain, dizziness, neck and left arm pain. He had no other significant past medical history.

The patient had initially visited his family doctor after experiencing shortness of breath while climbing a flight of stairs. When the electrocardiogram (ECG) showed two ventricular premature beats, the patient was referred to a cardiologist who prescribed calcium-channel blockers and ß-adrenergic blockers. The patient then began to experience chest pain, dizziness and left arm and neck pain which persisted even after medications were discontinued.

Although a work-up for chest pain revealed only a hiatus hernia, the patient became convinced that his chest pain was cardiac and that his other complaints were due to an as yet undetected disease. He began to keep a diary noting his pulse rate, abnormal sensations, dizziness, food and water intake as well as all bowel movements and urinations. He also presented twice to the emergency department and visited an otolaryngologist for investigation of vertigo. He was finally referred to T.G.H. where doctors thoroughly investigated his complaints but again no organic basis for them was found.

The patient was reluctant to be seen by Psychiatry. During the consultation he was angry and insulting, preoccupied with his symptoms and distrustful of the care he was receiving in hospital. Nevertheless, he would not admit to any negative feelings and he denied ever having felt depressed or anxious. He reported no past history of psychotic symptoms, no alcohol or drug addiction, no self harm, suicidal or homicidal tendencies. As well, the patient denied having recent stressors, fears of aging or experiences with anyone with heart disease.

### Discussion

The psychiatric diagnosis in this case was hypochondriasis. The patient was preoccupied with having a disease, apparently misinterpreting the symptoms of hiatus hernia and the physiological effects of anxiety as signs of serious organic pathology. The differential diagnosis included anxiety disorder and a possible somatoform pain disorder or conversion disorder, with arm and neck pain mimicking the symptoms of a myocardial infarct.

The patient seemed predisposed to somatization and hypochondriasis. He had alexithymic traits – being unable to experience or express affect – and probably directly channelled psychic distress into somatic symptoms such as pain. The patient also had some characteristics of an obsessive-compulsive personality with rumination about bodily complaints. Although he denied fears of aging, reaching "pension age" in the past year may have served as a reminder of his mortality, leading to the "medicalization" of his relatively benign symptoms.

Management consisted of reassurance that stress and anxiety rather than organic disease produced his symptoms. He was also encouraged to resume as normal a life as he could as soon as possible.

## Case 3

A 29 year old female paraplegic and former nursing aide presented with flank pain and gross hematuria. She was admitted to T.G.H. and treated with a diagnosis of pyelonephritis and cystitis. Cystoscopy revealed haemorrhagic cystitis.

The patient reported that her paraplegia was the result of a motor vehicle accident (MVA) in 1984. She said she was left with no movement in her legs and no voluntary bowel and urinary function. She also reported a history of chronic pancreatitis, parenchymal kidney disease (both unconfirmed) and a gall bladder operation two months previously.

In hospital the patient reported numerous unexplained aches and pains (abdomen, chest, back, neck). These were investigated with ECG, CT scan, ventilation/perfusion (V/Q) pulmonary scan and miscellaneous ultrasounds. No physical basis was found for her symptoms and psychiatry was consulted to investigate a possible psychogenic component to her pain. At this time, the medical team noted that there was no atrophy in her leg muscles as would be expected with paraplegia of five years duration. Evoked potentials suggested that there was more nerve and muscle function than she admitted to or believed. The medical staff felt manipulated by this patient and became increasingly irritated by her.

The patient reported that she was unhappy with the care she was receiving from her physician. She blamed him for not discovering what was wrong with her and prayed to God that he would be cursed. She believed that kidney disease or a stone was responsible for her pain and she wanted further investigations. She claimed that she knew her rights as a patient and was entitled to a second opinion.

The patient felt that her unresolved medical problems

were interfering with her high school studies and future career plans. She was forced to postpone writing a final exam because of her current illness and hospitalization. Similarly, in the previous year, she could not write an exam because of gastrointestinal troubles and gall bladder investigations.

When asked about personal and family history, the patient reported being physically abused as a child by her mother. As an adolescent she said she never got involved with what most teenagers like to do (music, parties). She always sat at home and studied and never dated. She also felt she needed a career before marriage so that "men would not take advantage of her".

On mental status exam the patient complained of severe pain but frequently smiled and giggled. There was no evidence of a thought disorder, her sensorium was clear though she refused to have her memory tested. The patient seemed frustrated throughout the examination.

She initially denied any past psychiatric history but eventually admitted to a two year history of depression after her accident. She described this period as a "living hell" consisting of sleep disturbances, anhedonia, tearfulness, decreased appetite and weight loss. She said that she briefly saw a psychiatrist and social worker during this time and was prescribed a benzodiazepine and an antidepressant. When the depression finally lifted, the patient reported that she was left with upper back pain.

### Discussion

In this case the patient exhibited aspects of several somatoform disorders. The differential diagnosis included factitious disease, malingering, conversion disorder, somatoform pain disorder, and a major depression. However, the apparent voluntary production of her paraplegia was felt to be most consistent with a factitious disorder. As well, her bladder infection might have been self induced. The patient also had the typical characteristics for factitious disease; being unmarried, having a work history in the health care profession and having a lack of close warm relationships.[5]

This patient reported that she had catheterized her bladder, perhaps unnecessarily, for the past five years. The need to be ill, to injure or to arouse herself might have been motivating factors. The patient had been physically abused as a child by her mother and there may also have been the wish to take revenge and antagonize or frustrate physicians as surrogate parental figures. Finally, there may also have been hypochondriacal fears that led her to produce symptoms or signs to convince her doctors to continue medical investigations.

Malingering was a possible diagnosis in that the patient might have used her "paraplegia" to obtain a financial compensation after her MVA.

The patient may also have experienced a conversion paralysis after her MVA as well as psychogenic aches and pains while in hospital. Being disabled and ill may have helped to relieve her of internal conflict and permitted her to avoid relationships as well as work and school responsibilities.

The patient's symptoms may also be partly explained by depression. She reported a past history of depression after her MVA and admitted that when the depression seemed to have lifted she was still "left with" upper back pain.

Management consisted of raising the possibility with her that stress might be worsening her somatic symptoms and informing her that tests suggested she should have much more physical ability. Admission to an in-patient rehabilitation program was recommended. Discussion of the nature of her psychiatric disorder helped to diminish the medical team's anger and feelings of manipulation.

However, the patient refused to accept an emotional explanation for her symptoms. When discharge was suggested she threatened suicide "so that the autopsy would show the doctors (she was) really sick". She did not accept admission into a rehabilitation program and left hospital. At last report she had presented to another downtown teaching hospital.

## Conclusion

Somatization involves the tendency to experience and communicate psychological distress in the form of physical symptoms. It is not necessarily pathological but can be a major feature of a variety of somatoform and psychiatric disorders. The somatizing patient will often present with aspects of several disorders; medical and psychiatric problems are not mutually exclusive.

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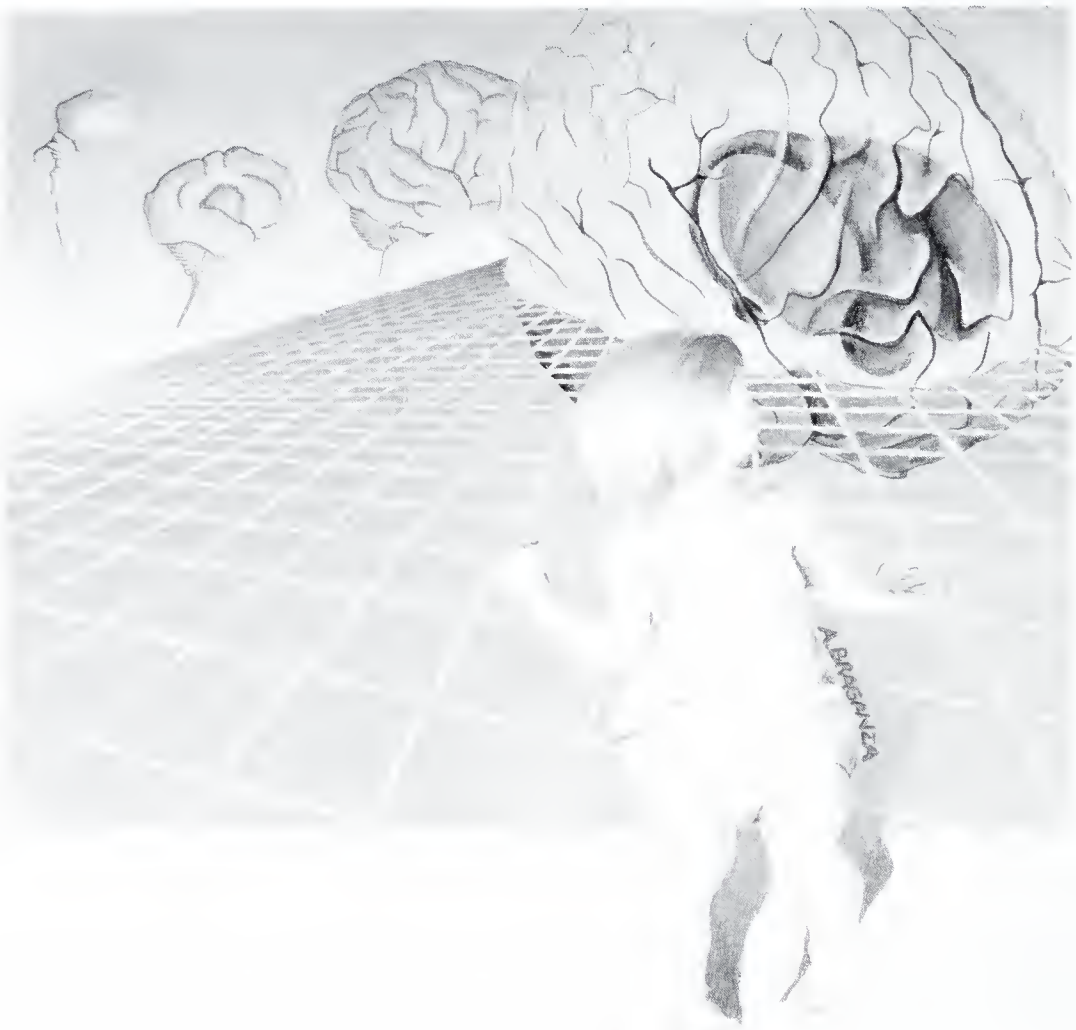

# Gait Analysis in Patients with Cerebral Palsy

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## Abstrait

Les enfants atteints de paralysie cérébrale exhibent une diversité d'évolutions caractéristiques de marche. Il existe présentement des interventions thérapeutiques, orthétiques et chirurgicales capables d'améliorer ces anomalies de démarche. Le traitement chirurgical offre des résultats inconsistants. L'essence du déficit neurologique de la paralysie cérébrale nous empêche de se servir de l'observation clinique pour découvrir l'étiologie précise de la démarche anormale. L'analyse de la démarche au laboratoire dirige les décisions chirurgicales en examinant la corrélation entre l'activité dynamique des muscles et les mouvements articulaires. Cette revue examine les méthodes variées d'analyser la démarche et leur utilité en ce qui concerne l'évaluation des patients atteints de paralysie cérébrale. Les facteurs musculosquelettiques ainsi que les stratégies du jour pour le traitement d'entretien seront aussi discutés. Il est nécessaire que les professionnels de la santé soient mis au courant de l'analyse au laboratoire afin d'améliorer le traitement des patients exhibant des démarches anormales.

## Abstract

Children with cerebral palsy exhibit a variety of abnormalities in their walking patterns. Therapeutic, orthotic, or surgical interventions can be used to improve a child's gait. An inconsistent outcome has been the hallmark of surgical treatment of these patients. Due to the nature of the neurological deficit in cerebral palsy, it is often inadequate to use clinical observation to identify the exact etiology of the abnormal gait pattern. Laboratory gait analysis leads to better operative decisions through the correlation of dynamic muscle activity and joint motions. In this review, the methods of gait analysis are outlined with reference to their use in the assessment of patients with cerebral palsy. Musculoskeletal factors that contribute to abnormal gait patterns are discussed along with current management strategies. The management of patients with abnormal gait patterns could be improved if more health professionals became aware of the benefits of gait analysis.

## Introduction

Cerebral Palsy (CP) is the most common chronic disabling childhood condition in North America with an incidence of 1-5/1000 live births, and a prevalence of 400,000.[1] Cerebral Palsy is a disorder of movement and posture caused by a non-progressive brain lesion affecting the immature brain, which may occur pre-natally, peri-natally or post-natally. These children show a delay with motor and cognitive developmental milestones. The many associated medical problems include: ocular abnormalities, hearing loss, seizure disorders, scoliosis, hip dislocation, drooling, perceptual disorders, and communication difficulties.

The main types of cerebral palsy are spastic, athetoid, ataxic, or mixed. Spastic cerebral palsied children may have hemiplegic, diplegic, or quadriplegic involvement. Spastic patients are characterized by co-contraction of agonist and antagonist muscle groups, exaggerated stretch reflexes, and hyperreflexia. Athetoid patients have involuntary gross writhing movements in the extremities and hyporeflexia. Ataxic patients are characterized by decreased coordination and equilibrium. If mobile they will exhibit a wide based staggering gait. Although all types of cerebral palsied children have characteristic gait patterns, laboratory gait analysis centres on the spastic children since they undergo the majority of surgical interventions.

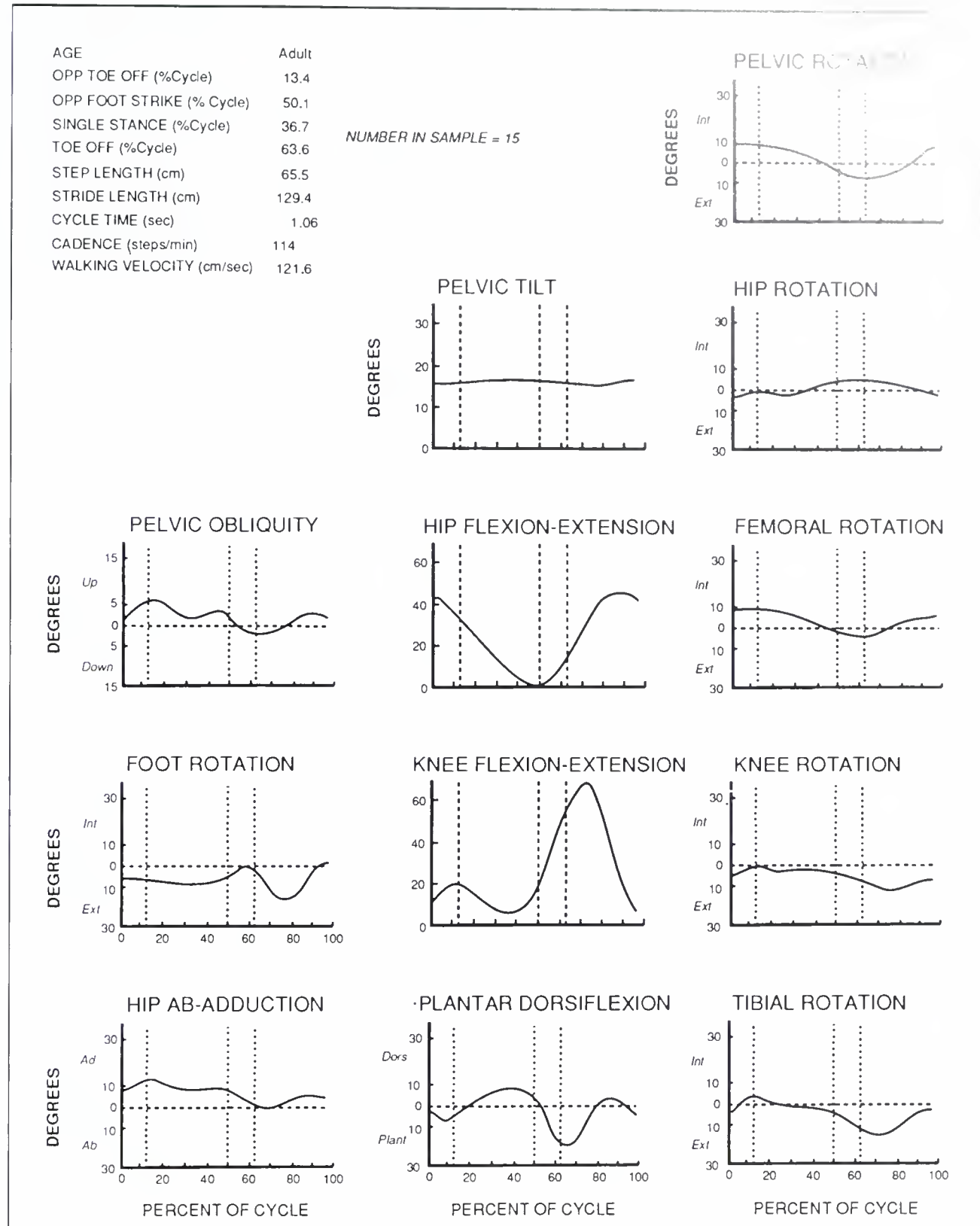


**Figure 1:** Normal Kinematic Data: Mean values of pelvic, hip, knee, and ankle motion for 15 adults.
The abscissa is percentage of gait cycle and the ordinate is degrees of motion. The vertical dotted lines mark intervals representing different phases of the gait cycle: stance phase (SP) double support, SP single support, SP double support and swing phase consecutively.

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The etiology of CP has changed over the last 25 years. Prior to 1950, dystocia, encephalitis and erythroblastosis were common causes. Erythroblastosis was associated with the development of kernicterus, which showed a strong correlation with the development of athetoid CP. Current management of Rh incompatibility has resulted in a decreased incidence of erythroblastosis. Modern technological advances in neonatology have resulted in increased survival in preterm infants. Consequently, prematurity has become recognized as a major contributing factor to the development of spastic diplegia.[2]

One of the primary questions of parents is, "Will my child walk?". The ability to stand supported or to perform the stepping response when held in an upright posture are often misinterpreted as early signs leading to walking. Poor prognosis of walking has been demonstrated by:

1 the inability to sit alone by age 4 years,[3]

2 persistence of infantile reflex activity beyond 18 months,[1] and

3 the absence of developmental reflexes.[2]

The acquisition of postural control against gravity is necessary for the development of successful walking, thus, standing equilibrium tests are helpful for determining a walking prognosis.[4] A child with poor equilibrium in all

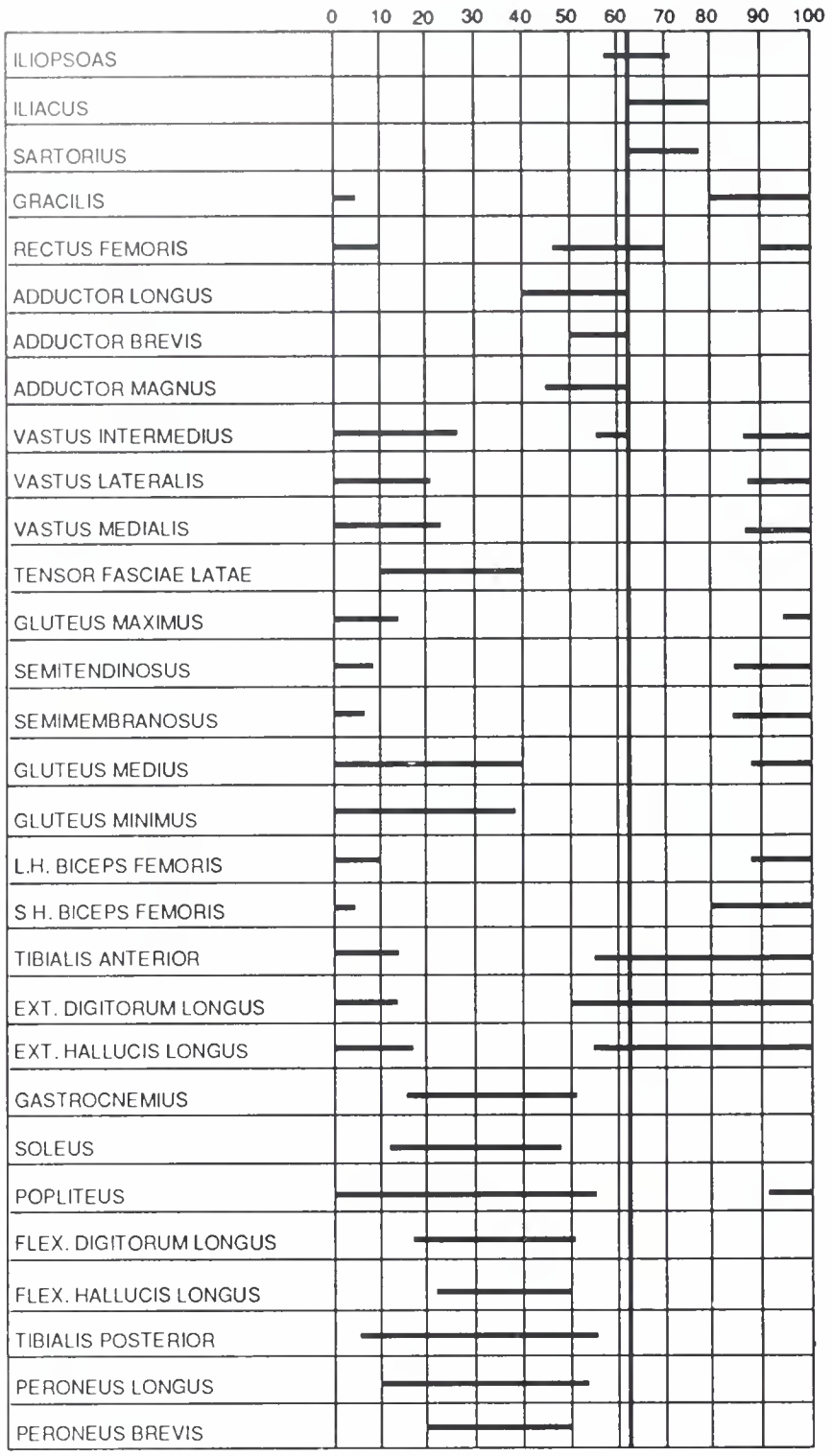


**Figure 2:** Normal EMG Data: Averaged adult muscle phasic activity during a gait cycle.Percentage of gait cycle is marked along the horizontal. The vertical line separates stance and swing phase respectively.

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four planes will need a 4-legged walker, whereas a child with poor anterior-posterior equilibrium may need forearm crutches.

## Gait Laboratory

The gait laboratory is still used primarily as a research tool in Canada. The success or failure of such a lab depends on finances, space, trained personnel and the attitudes of the medical profession.[5] Information obtained from a gait lab can be divided into four categories:

1 basic temporal and stride data
2 kinematic descriptors
3 kinetic analysis, and
4 electromyographic data.

Temporal and stride information can be obtained from film data with digital displays or foot switches. Typical foot switch positions are the heel, 5th metatarsalphalangeal joint, 1st metatarsalphalangeal joint, and the great toe. Foot switches demonstrate foot contact patterns. Stride time is the temporal interval between right heel strike to subsequent right heel strike. Right stance time is the temporal interval between right heel strike and right toe-off. Right swing time is the interval between right toe-off and right heel strike. Stance time is approximately 60% of stride time in normals. Stance may be further divided into single support, occurring during mid-stance, and double support, occurring in early and late stance. Stride length, right or left step lengths, velocity (metres/second), and cadence (steps/min) can also be calculated with this data.

Kinematics refer to the description of joint motions (angles, velocities, and accelerations) as a function of time and space. This information can be obtained from cinematography, goniometry, and television systems. Many new motion analysis systems use optoelectrics in combination with computer systems and are capable of describing joint motion in three-dimensions. Each graph in Fig. 1 describes the motion of a joint in a specific plane during the four phases of a gait cycle. Kinematic descriptors provide quantitative data on joint motions, but give little information on the neurological and biomechanical mechanisms that cause the atypical pattern.

Kinetic analysis reveals biomechanical information invisible to the eye. With the use of force plates, ground reaction forces can be determined in three planes: vertical, medial-lateral shear and foreward-after shear. These forces can be used to calculate the changing location of the centre of pressure and external joint torques during stance. The centre of pressure can be defined as the point of origin of the resultant ground reaction force vector. External joint torques can be calculated from the resultant ground reaction forces and joint centre locations.

Electromyography (EMG) analysis is performed with surface or indwelling electrodes which record the activity of muscle groups or individual muscles, respectively. EMG data is correlated with temporal data from foot switches. The main value of the data is to reveal the PHASIC activity of the muscles that accelerate or decelerate the limbs and stabilize the joints. Fig. 2 demonstrates the phasic electrical activity of the many muscles that are active during gait.

## Clinical Observation of Cerebral Palsy Gait

Many abnormal gait patterns can be identified through clinical observation by a trained eye. The motion of the pelvis, hips, knees and feet, as well as the spatial relationships during stance and swing, must be analyzed individually.[2] "Toe-walking" is the most common problem seen with spastic CP patients. It is difficult to determine clinically whether the dynamic equinus deformity is due to overactive tricep surae or hamstrings. Overactive hamstrings will bring a patient's centre of gravity posteriorly and compensation will be made by toe-walking. A "stiff knee gait" is characterized by difficulty with foot clearance during swing. This gait pattern may be due to primary

overactivity of the quadriceps or failure to recognize hamstring/quadricep co-contraction clinically before surgical hamstring lengthening. A "toe-in" gait is often due to internal rotation of the entire lower extremity.

## Laboratory Analysis of Cerebral Palsy Gait

Gait laboratory analysis can be used for research, pre-operative decisions, post-operative monitoring or decisions regarding orthotics, casting and therapy. Many treatment plans suffer due to inadequate analysis. Two patients may appear identical clinically, undergo similar surgical procedures, yet demonstrate completely different outcomes. For example, the correction of varus foot deformities with tibialis posterior lengthening will depend on the extent of involvement of other muscles. The lab analysis is useful in differentiating between primary and secondary abnormalities of posture and gait.[2,6] Identifying the exact cause of a dynamic deformity is essential to ensure the best possible outcome for treatment.

EMG is the most widely used assessment tool for CP patients since electrical muscle activity is a primary indicator of the final neural control pattern.[5] Analyzing muscle activity is useful for pre-operative decisions since the state of activity of the muscles cannot be predicted reliably from inspection.[7,8] Perry et al. (1988) demonstrated that EMG could be used to identify the deforming muscle and its pattern of activity in varus deformities of the foot in CP.[8] The EMG and foot switch pattern of a patient in this study is shown in Fig. 3. The activity of the tibialis anterior and posterior is correlated with phases of the gait cycle through the use of foot switches. The tibialis anterior contributed to the varus deformity in 75% of the cases and the tibialis posterior in 54%. They felt that better results could be obtained by basing operative procedures on EMG data. Continuous electrical activity of a muscle is an indication for surgical lengthening and out of phase activity is an indication for a transfer. Lengthening of muscles overcomes the spastic overactivity of shortened muscles. Transferring a muscle removes a deforming force and provides support to replace a muscle that is weakened or inactive.[7]

Two post-operative complications are the development of crouch gait after rectus femoris release and knee hyperextension following hamstring lengthening. It is important to analyze the state of activity of both the quadriceps and hamstrings prior to surgery. Spastic quadriceps generally accompany spastic hamstrings and can be seen by prolonged and/or out of phase EMG activity.[9] EMG does not appear to be useful for post-operative assessment since the phasic activity of the muscles does not change to any significant extent.[10]

Kinematic motion analysis is useful for quantitative pre- and post-operative measurements. Sutherland (1975) used pre- and post-operative motion analysis on patients with stiff-knee gait associated with rectus femoris overactivity. His study revealed an improvement in early swing phase knee flexion after rectus femoris release.[11] Gage et al. (1984) demonstrated a statistically significant increase in stance knee extension and swing ankle dorsiflexion ($p = 0.05$ and 0.002, respectively) in spastic diplegics post-operatively.[10] The surgical procedures for each patient varied and were based on the pre-operative gait analysis. The outcome of the surgical interventions is represented graphically in Fig 4.

The use of force plates is limited in CP children due to their difficulty with taking a single step on the plate which is required for proper data collection. Sutherland and Cooper (1977) used force plate data to explain the pathomechanics of progressive crouch gait in spastic diplegics. They revealed the cause to be excessive stance phase knee flexion torques.[12]

Temporal and stride parameters have been used to compare CP gait to that of normals. Velocity and stride length are generally decreased. Stance time as a percentage of stride time is usually higher than in normals. This is due to decreased single limb stability. CP children may increase their velocity and stride length post-operatively, but their cadence remains constant. This suggests that the increased stride length is the cause of the increased velocity.[13]

**Figure 3:** Foot switch and EMG pattern of a patient with a varus foot deformity. Foot switch data outlines the stance and swing phases of gait, ie. switches are on during stance. This patient demonstrates an abnormal EMG pattern shown by continuous tibialis anterior activity and swing phase tibialis posterior activity. Refer to fig. 2 for normal muscle activity.

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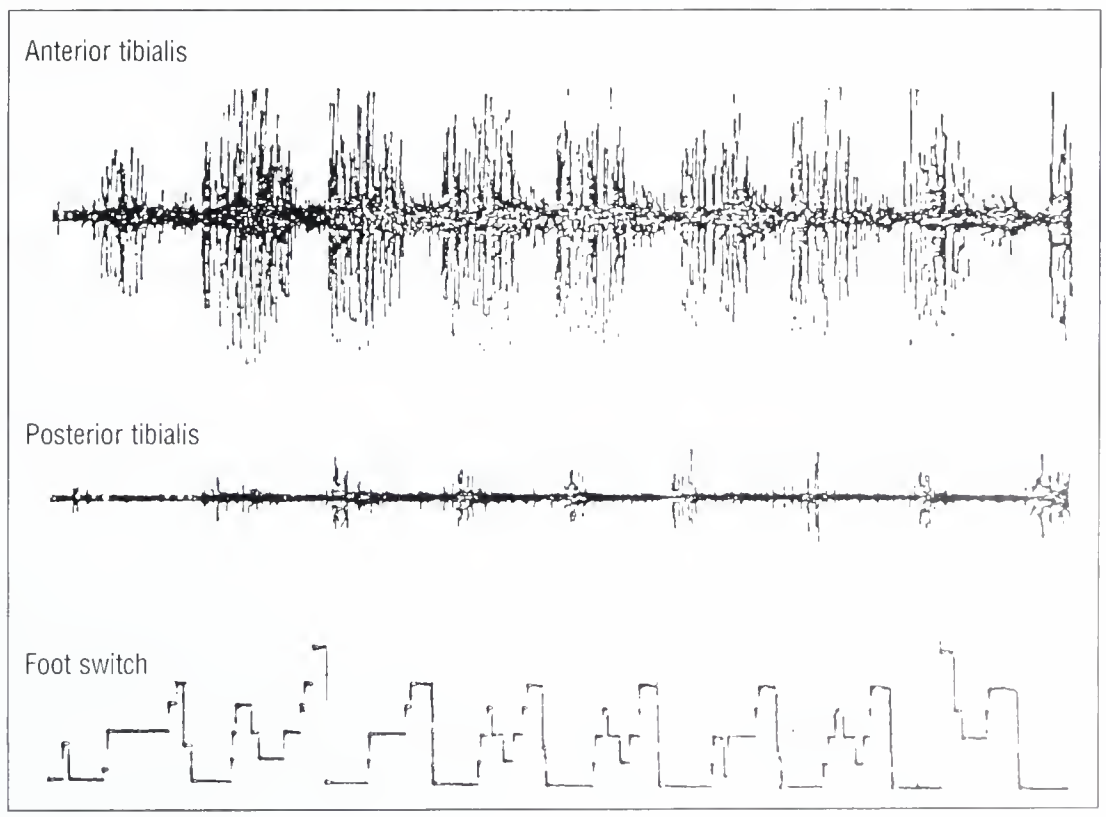


## Spastic Diplegics

Seventy-nine percent of spastic diplegics can walk independently, while 13% will walk with crutches, and 7% with rollator walkers.[14] The average age of onset of walking is 48 months with a functional gait peak at approximately six years of age. Lower limb involvement in spastic diplegia includes: hip flexion

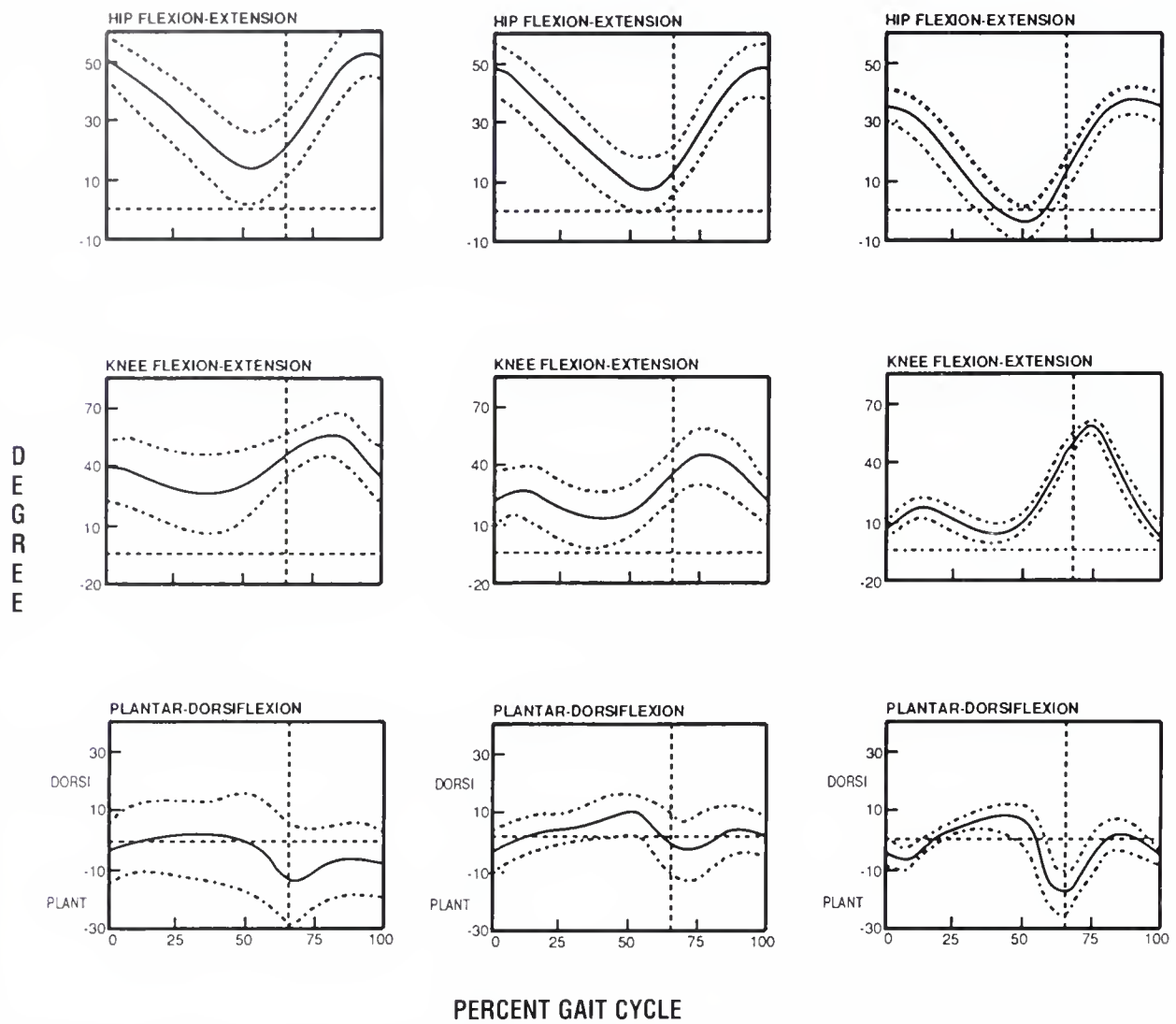


**Figure 4:** Average and SD for pre-operative (left), post-operative (middle), and normal kinematic data (right). Stance knee extension and swing ankle dorsiflexion show a post-operative improvement.

Reproduced with permission from *Pre and Post-Operative Analysis in Patients with Spastic Diplegia: a preliminary report.* J Pediatric Orthop. Gage et al; 1984; 4:p715-725.

contractures, hip adductor contractures, hip internal rotation, knee flexion or extension patterns, ankle equinus and ankle varus/valgus deformities.

The hip flexion contracture may be caused by overactivity of the rectus femoris, but more commonly is due to overactivity of the iliopsoas. When surgical intervention is considered it is important to preserve the activity of the iliopsoas since this is the main hip flexor responsible for clearing the foot during early swing. For this reason, an iliopsoas recession is beneficial since this operation does not cause overweakening.

A spastic diplegic with a hip adductor contracture will be noted to have close approximation of the knees and thighs, and if severe enough, scissoring of the legs will result. The goal of treatment for overactive adductors is to increase the base of support to normal limits (5-10 cm). Surgery may include adductor tenotomies, myotomies or anterior branch obturator neurectomies, although the latter is out of favour. Post-operative complications include a wide based gait or permanent "frog leg".

Hip internal rotation is observed clinically as "toe-in" gait. The internal rotation may be caused by overactive muscles or femoral anteversion. The semitendinosus and the anterior portion of the gluteus medius have been implicated as muscles that contribute to hip internal rotation.[14] Femoral anteversion can be suspected clinically if hip internal rotation is >60 degrees and hip external rotation <30 degrees in full knee extension. Dynamic internal rotation results because the greater trochanter must rotate out to allow the gluteus medius to contract efficiently for lateral stability during stance. A derotational femoral osteotomy is a common operative procedure used to improve femoral anteversion.

A dynamic knee flexion deformity may be the result of overactive hamstrings or weakened quadriceps. It is important to monitor the motion and activity around the hip and ankle as well, to determine their contribution to the gait pattern. If a hip flexion contracture is ignored and hamstring lengthening performed, the post-operative gait may be characterized by excessive lumbar lordosis. If a fixed ankle equinus deformity is ignored and hamstring lengthening performed, post-operative anterior instability may lead to a previous independent walker requiring crutches. Surgery may include hamstring tenotomies, transfers, or lengthening, the latter being the most common. Lengthening often excludes the bicep femoris which is responsible for the initiation of knee flexion during swing.

Dynamic ankle equinus may be the result of overactivity of the tricep surae or a compensatory response to hamstring spasticity in an attempt to bring the centre of gravity anterior to the ankles. Ankle valgus appears to be more common in spastic diplegics than varus.[14] Valgus may be due to peroneal overactivity or tibialis posterior underactivity. Surgery includes tenotomies and transfers. In advanced cases subtalar or triple arthrodeses may be performed.

## Spastic Hemiplegics

Most spastic hemiplegics can walk by 18-21 months of age. The lower limb involvement is usually concentrated at the ankle with equinus and varus deformities. Equinus deformities prevent heel contact and result in toe-walking. Attempts at improving equinus have included myoneural blocks, casts, and orthotics. The main surgical intervention

is Achilles tendon lengthening. Uncorrected equinus may lead to painful callosities at the metatarsalphalangeal joints, an unstable talus and lumbar lordosis due to compensation for the forward shift of the centre of gravity.[1,14] Ankle varus is usually caused by increased activity of the tibialis anterior and tibialis posterior but the soleus, flexor digitorum longus, and flexor hallucis may also contribute to this deformity. In the past, surgical intervention was based on whether the varus deformity was seen during stance, indicating tibialis posterior overactivity, or swing, indicating tibialis anterior overactivity. Better operative decisions can be made when the specific deforming muscle(s) can be properly identified. Spastic hemiplegics may also exhibit a leg length discrepancy. If severe, this may be corrected by epiphyseal arrest of the normal limb or by using various procedures to lengthen the shortened limb.

In conclusion, gait analysis is a beneficial tool for the investigation of cerebral palsy patients who are candidates for corrective surgery.

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# Acute Pulmonary Toxicity of Aerosolized Pentamidine in HIV-Seropositive Patients

*Karl T. Iglar 9T1, Martin A. Katzman B.Sc. 9T1, William M. Meade 9T1, and Charles K. Chan M.D. F.R.C.P.; in collaboration with Toronto Aerosolized Pentamidine Study (TAPS) Group from the Department of Medicine, The Wellesley Hospital, University of Toronto, Toronto, Canada*

## Abstrait

**Entre les mois de juin 1988 et février 1989, 36 patients furent inscrits pour un essai à l'aveuglette double; tous les participants étaient séropositifs pour le VIH et tous avaient conquis un épisode de pneumonie Pneumocystis carinii (PCP). Le but était d'évaluer la toxicité du pentamidine aérosol (PA) comme prophylaxie contre les épisodes subséquents de PCP. Chaque patient fut sujet à la spirométrie avant et après l'administration du PA. Les résultats de base des épreuves de fonctions pulmonaires étaient comparables entre les deux groupes. Onze (65%) des patients qui avaient reçu le PA développèrent une toux tandis que quatre (21%) démontrèrent une réduction de débit pulmonaire modeste mais importante (avant traitement, $FEV_1$/FVC moyenne de 80%; après traitement, $FEV_1$/FVC moyenne de 72%). Cette réduction de débit était causée par le bronchospasme. Par contre, quatre (21%) des patients du groupe placebo développèrent une toux mais leurs débits pulmonaires sont demeurés comparables (avant traitement, $FEV_1$/FVC moyenne de 85%; après traitement $FEV_1$/FVC moyenne de 86%). Toutes les épisodes de bronchospasme étaient spontanément résolutifs et sensibles au salbutamol inhalé. En conclusion le pentamidine entraîne quelquefois des épisodes de toux mais, les épreuves de fonctions pulmonaires démontrent que l'incidence du bronchospasme modéré est beaucoup plus basse.**

## Abstract

**From June 1988 to February 1989, 36 HIV-seropositive patients who had experienced one previous episode of Pneumocystis carinii pneumonia (PCP) were enrolled in a randomized double-blinded placebo-controlled trial to assess the toxicity of aerosolized pentamidine as prophylaxis against a second episode of PCP. Each patient underwent spirometry before and after each aerosolized treatment. There was no significant difference in the results of baseline pulmonary function testing between the aerosolized pentamidine and the aerosolized saline groups. Eleven (65%) patients in the aerosolized pentamidine group developed cough, while four demonstrated a modest but significant reduction in the flow rates post-aerosolized pentamidine treatment (mean pre-treatment $FEV_1$/FVC was 80%, and mean post-treatment $FEV_1$/FVC was 72%) due to bronchospasm. Four (21%) patients in the placebo group developed cough, but no significant change in the flow rates was observed (mean pre-treatment $FEV_1$/FVC was 85%, and mean post-treatment $FEV_1$/FVC was 86%). All episodes of bronchospasm were self-limited and responded to inhaled salbutamol. We concluded that aerosolized pentamidine treatment is occasionally associated with coughing attacks (65%) but the actual incidence of bronchospasm as confirmed by pulmonary function testing is much lower (24%) and when it does occur, is generally quite mild.**

## Introduction

Pneumocystis carinii pneumonia (PCP) is the most common pulmonary infection in patients infected with human immunodeficiency virus (HIV). PCP develops in approximately 80% of HIV-seropositive patients.[1,2] The case fatality rate from this condition is 20%,[3] with a recurrence rate of 50% within the next year.[1,2]

Two pharmacologic agents, trimethoprim-sulfamethoxazole (TMP-SMX) and pentamidine, are presently used for PCP treatment. There is no significant difference in efficacy between these two medications.[4,5] Unfortunately, the routes of administration used, intravenous and oral for TMP-SMX[6] and intravenous and intramuscular for pentamidine,[4] result in severe systemic toxicities.[13]

In an attempt to decrease the incidence of adverse drug reactions and improve the efficacy of PCP therapy, aerosolized pentamidine (AP) has been widely investigated as an alternative for therapy and prophylaxis. AP has been shown to be effective in mild-to-moderate PCP,[7,8] and as a primary (prevent original infection) and secondary (prevent recurrance) prophylactic agent.[11,13] However, it is not completely free of complications. Those reported in previous case reports and uncontrolled descriptive studies include cough,[11] bronchospasm, and disseminated pneumocytosis,[7] pulmonary hemorrhage and spontaneous pneumothorax.[9-12]

We designed a randomized, placebo-controlled double-blinded study to compare the toxic effects of AP versus aerosolized saline on the bronchial tree. It was our intent to determine the incidence and assess the severity of acute pulmonary toxicity of pentamidine. To the best of our knowledge, this is the only controlled trial assessing AP toxicity.

Table I Demographic and Clinical Characteristics of Study Patients

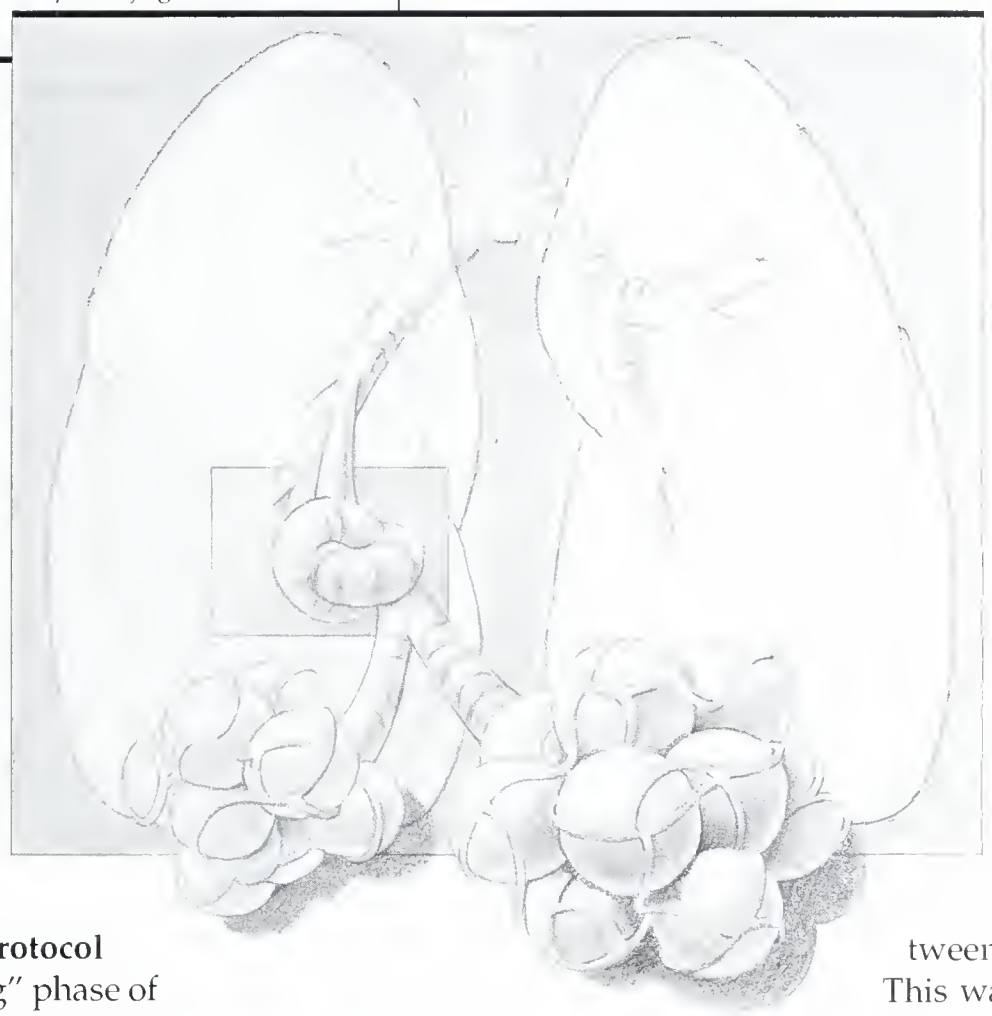
| Variables | Placebo Group (mean ± S.D.) | Pentamidine Group (mean ± S.D.) |
|---|---|---|
| N | 19 | 17 |
| *Age (yr) | 39.2 ± 9.2 | 34.0 ± 5.5 |
| Height (cm) | 175.4 ± 7.2 | 174.8 ± 9.0 |
| Weight (Kg) | 69.6 ± 10.4 | 66.6 ± 12.7 |
| Sex (M:F) | 18:1 | 16:1 |
| Risk Group | 17-Homo/Bisexual<br>2-Other | 16-Homo/Bisexual<br>1-Other |
| AZT (%) | 79 | 59 |
| Date of last episode of PCP (mo) | 3.8 ± 1.7 | 3.8 ± 1.8 |
| Date since diagnosis of HIV (mo) | 4.5 ± 2.7 | 3.9 ± 2.0 |

** There is no statistically significant difference between the two groups in all the variables, with the exception of age, where the P-value is 0.02.*

## Methods

### Patient Population

From June 1988 to February 1989, 36 HIV-seropositive patients with one previous episode of PCP were enrolled in a randomized placebo-controlled double-blinded study in order to evaluate the toxicity of AP for the prophylaxis of PCP. Random numbers generated from computerized sources were used for patient allocation. Nineteen patients were placed in the AP group and 17 patients in the placebo group.



### Aerosolized Pentamidine Protocol

As part of the initial "loading" phase of the AP prophylactic protocol, each patient received five aerosolized treatments (pentamidine or normal saline placebo) over a 2-week period every 2-3 days.

The patients underwent complete pulmonary function testing prior to entry into the study, and spirometric investigation was performed before and after each treatment in order to objectively assess changes in the patient's pulmonary function.

The patients were also interviewed before and after each treatment to ascertain subjective pulmonary symptoms (cough, dyspnea, chest tightness).

### Aerosolized Pentamidine Administration

One millilitre of normal saline was added to a vial containing either placebo or 60 mg of pentamidine (Fisons; Rochester, NY) which was then placed in a FISONeb nebulizer (Fisons; Rochester, NY). The patient, under supervision, inhaled the solution through the mouthpiece via a self administration process that took place over a fifteen to thirty minute period. The blinding code was not revealed until the end of the study.

### Pulmonary Function Testing

Pulmonary mechanics were measured by a computerized pulmonary function testing system (Collins 03000, W.E. Collins Inc., Braintree, Massachussetts).

### Data Analysis

Continuous variables such as pulmonary function test results, age, etc., were expressed as means and standard deviations, and the results of the two groups (placebo and pentamidine groups) were compared using Student's T-Test. Categoric variables, such as sex, were compared using Fisher's Exact Test. A P-value $< 0.05$ was considered as statistically significant.

## Results

Data on the efficacy of AP as a secondary prophylaxis is presented elsewhere.[13] This paper purely focuses on the acute pulmonary toxicity of this treatment.

Comparing the placebo group to the pentamidine group, both in terms of demographic characteristics (Table I) and baseline pulmonary function test results (Table II), the only significant difference between the two groups was age. This was probably of no clinical significance.

Table III and Figure 1 compare the incidence of adverse drug reactions between patients receiving aerosolized pentamidine and those receiving aerosolized placebo. Both treatments produced cough, but a significantly higher proportion of patients in the pentamidine group developed cough (65% versus 21%). Bronchospasm, which we defined as a reduction of $FEV_1/FVC > 5\%$ following treatment, did not occur in the placebo group but was noted in 24% of the pentamidine group. Bronchospasm was not detected in any of the patients who did not complain of cough.

With regard to the percentage change in $FEV_1$ after each of the five loading doses (Fig. 1) there was no apparent sensitization to AP or placebo during the loading period.

Table II Baseline Pulmonary Function Test Results

| Variables | Placebo Group* (mean ± S.D.) | Pentamidine Group* (mean ± S.D.) |
|---|---|---|
| N | 19 | 17 |
| TLC (L) | 6.2 ± 1.21 | 6.23 ± 1.25 |
| FVC (L) | 4.29 ± 0.80 | 4.40 ± 0.86 |
| $FEV_1$ (L) | 3.63 ± 0.69 | 3.71 ± 0.68 |
| $FEV_1$/FVC (%) | 84.53 ± 5.08 | 84.53 ± 5.66 |
| DLCO (ml/M/mmHg) | 26.06 ± 5.83 | 26.76 ± 5.87 |

TLC = Total lung capacity
FVC = Forced vital capacity
$FEV_1$ = Forced expiratory volume in 1 sec
DLCO = Diffusing lung capacity corrected for hemoglobin

** There is no statistically significant difference between the placebo and pentamidine groups in the baseline pulmonary variables measured.*

Table III Adverse Reactions to Aerosolized Pentamidine and Aerosolized Placebo

| Adverse | Placebo Group | Pentamidine Group | P-Value Reactions |
|---|---|---|---|
| N | 19 | 17 | |
| Cough | 4 (21%) | 11 (65%) | <0.01 |
| Bronchospasm* | 0 ( 0%) | 4 (24%) | 0.04 |

** Significant reduction of flow rates ($FEV_1$/FVC reduction > 5%)*

## Discussion

Since AP was first used for the treatment and prophylaxis of HIV-seropositve patients against PCP, a number of acute pulmonary adverse drug reactions have been observed. This randomized placebo-controlled double-blinded study on a cohort of 36 patients has allowed us to draw a number of conclusions regarding the acute pulmonary toxicity AP.

First, patients receiving any aerosolized substance (placebo or pentamidine) show a propensity to develop a cough, but coughing occurs with a much higher frequency in patients receiving AP, with some 65% of the patients complaining of cough. Second, only patients on AP developed bronchospasm, indicated by a 12-21% drop in the $FEV_1$. Bronchospasm was only noted in those patients who also experienced cough. Cough and bronchospasm were relieved by the administration of ß2-adrenergic agonist, which is now routinely used in our clinic prior to AP treatment in order to decrease the risk and degree of cough or bronchospasm. As such, in a limited health care environment where regular spirometric monitoring is not readily available, it may be prudent to treat all AP patients who are experiencing cough with ß2-adrenergic agonist in order to minimize unpleasant acute pulmonary toxicity.

Third, neither sensitization nor desensitization marked by an increase or decrease in the amount of coughing or bronchospasm was noted over the administration of five AP treatments in this study.

It is important to note that the results of this study are only applicable to treatment using the FISONeb nebulizer with the dosage regimen described, and should not be generalized to other AP protocols.

In conclusion, it is readily apparent from the results of this study that AP used for prophylaxis of PCP is generally well tolerated. The high frequency of cough and bronchospasm, should not be of great concern as these can easily be relieved by symptomatic therapy. However, long-term follow-up studies on a larger cohort of HIV-seropositive patients receiving regular AP treatment should be undertaken to identify factors related to the development of acute pulmonary toxicity and further evaluate the long-term pulmonary effects associated with AP.


## Acknowledgement

The authors wish to thank Mrs. Fehmida Nurmohamed for preparing the manuscript.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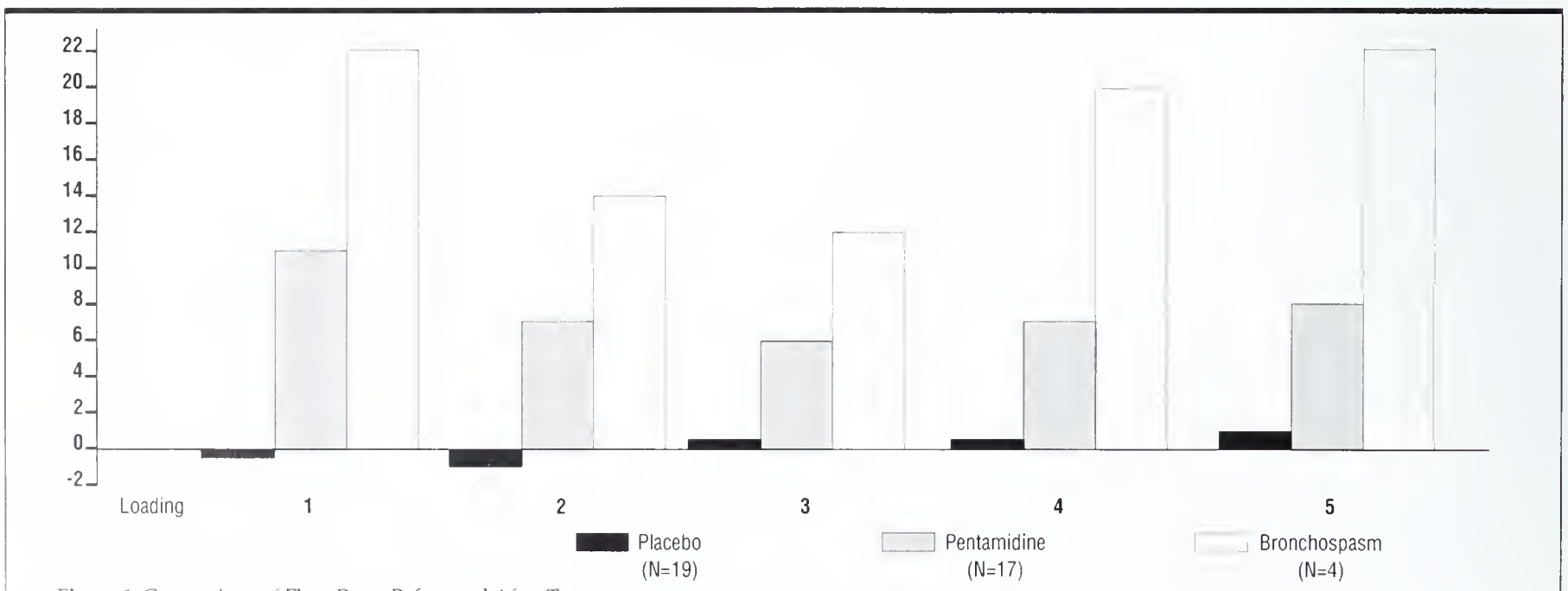


Figure 1: Comparison of Flow Rates Before and After Treatment

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# A 37 Year Old Man with Generalized Edema

*Osborne I. Noronha 9T1, Phillip J. Porter 9T1, and Edward H. Cole, M.D. F.R.C.P.*

## Case Report

Mr. L.M. is a 37 year old white truck driver admitted to hospital for investigation of swelling of the hands, trunk, buttocks and feet of eight days duration.

He was in good health until a year ago, at which time he experienced two weeks of nausea, vomiting, occasional diarrhea, fever, chills and colicky abdominal pain. Immediately following this illness he developed penile edema, which has fluctuated over the last year but has not yet resolved. One month prior to admission he again suffered from nausea. In addition, he vomited bile-stained food particles and had three to four watery, yellow-green bowel movements per day mixed with fresh blood. Two weeks later he noticed the onset of swelling and erythema involving the whole body and it was at this time that he sought medical advice.

Past and current medical histories as well as family and social histories were otherwise unremarkable. Positive findings on functional inquiry included a 30 pound weight gain over the last year, chronic fatigue and nocturia once nightly.

On physical examination, Mr. L.M. appeared his stated age and was in no apparent distress, but appeared unwell and had 3+ generalized pitting edema. He was afebrile, with a blood pressure of 110/70 (no postural drop) and a regular pulse of 80/min. The jugular venous pulse (JVP) was 1 cm below the sternal angle. The abdomen was soft and non-tender with no organomegaly and there was moderate ascites. The rest of the physical examination was unremarkable.

The results of the relevant investigations were as follows (normal values in brackets):

- Hb 182 g/L(140-180 g/L),
- WBC 10.5 x $10^9$/L(4-11 x $10^9$/L),
- Cholesterol 8.86 mmol/L(<5.2 mmol/L),
- TG 11.90 mmol/L(<2.3 mmol/l),
- Albumin 21 g/L(40-60g/L),
- Total protein 47 g/L(60-80 g/L),
- $Ca^{2+}$ 1.99 mmol/L (2.20-2.60 mmol/L), and
- $Na^+$ 130 mmol/L(137-143 mmol/L).

His CXR, upper GI series, abdominal ultrasound, renal function tests, urinalysis, liver function tests, liver enzymes, stool culture, alpha-one antitrypsin and bone marrow were all normal. An echocardiogram revealed a trivial pericardial effusion.

**Table I Differential Diagnosis of Edema**

| | |
|---|---|
| *A* | ***Localized*** |
| | Lymphatic Obstruction |
| | Venous Obstruction |
| | Increased Capillary Permeability |
| *B* | ***Generalized*** |
| | Cardiac eg. congestive heart failure, pericardial effusion |
| | Renal eg. nephrotic syndrome, chronic renal failure |
| | Hepatic eg. hypoalbuminemia |
| | Gastrointestinal eg. protein losing enteropathy, malnutrition |
| | Vascular eg. vasculitis |

## Pathophysiology

This case presents a puzzling example of a common clinical problem – edema (see Table I). In order to develop a rational approach to the management of edema it is important to first understand the physiologic parameters which, when altered, can lead to its formation. Edema is swelling resulting from an increase in the interstitial fluid volume. If generalized, an increase in volume of two to three litres is often required before becoming clinically apparent. It will be recalled that the interstitial space is one component of the extracellular fluid volume (ECFV); the other compartment being the vascular or plasma volume. The distribution of fluid between these two compartments of the ECFV depends on the Starling forces; that is, the balance of the hydrostatic and oncotic pressures. This can be expressed by Starling's Law which states that net filtration = capillary permeability x (hydrostatic pressure gradient – oncotic pressure gradient). Under normal conditions, the factors promoting movement of fluid out of the vascular system (capillary hydrostatic pressure and interstitial oncotic pressure) are almost, but not quite, balanced by the forces promoting movement in the opposite direction. The net result is a slight gradient favouring filtration towards the interstitium. A steady state is normally maintained by returning an amount of fluid equivalent to that filtered to the venous system via the lymphatics.

Normally the kidney minimizes changes in ECFV and therefore prevents edema by appropriately regulating sodium excretion. However, in certain pathological states this tight regulation can be disrupted, with the resultant failure to excrete ingested salt leading to increases in ECFV. In some cases the salt retention is due to abnormal renal function (primary salt retaining states, such as in the nephritic syndrome) while in others the kidney is responding to stimuli falsely interpreted as ECFV depletion (secondary salt retaining states such as in congestive heart failure). While ECFV is increased in both conditions, the essential difference is that effective circulating

arterial volume is increased in primary but decreased in secondary salt retention states.

In both cases, the development of edema is the result of alterations in one or more of the forces controlling fluid movement at the capillary level, the alteration being in a direction that encourages movement into the interstitium. These forces can include increased capillary hydrostatic pressure, decreased plasma oncotic pressure, and enhanced capillary permeability. Thus in renal insufficiency the salt retention leads to increased hydrostatic pressure and edema. In other situations there is a primary abnormality at the capillary level, such as decreased oncotic pressure. This causes a reduced effective circulating volume, which is interpreted by the kidney as volume depletion, and thus leads to an attempted secondary renal compensation by salt retention. In the course of returning the plasma volume towards normal, there is further fluid loss to the interstitium because the underlying capillary abnormality still exists.

Edema can be classified as localized or generalized. Localized edema is the result of venous or lymphatic obstruction or increased capillary permeability. Generalized edema can be due to increased capillary hydrostatic pressure, decreased plasma oncotic pressure, or rarely, diffusely increased capillary permeability. The systems which are important in the pathogenesis of generalized edema are the cardiovascular, gastrointestinal, hepatic, and renal systems. In cardiac causes such as heart failure and constrictive pericarditis, as well as in renal insufficiency, the operative mechanism is increased capillary hydrostatic pressure. Plasma oncotic pressure is the problem in starvation or malabsorption syndromes (decreased protein intake); the nephrotic syndrome and protein-losing enteropathies (increased losses); and liver disease (inadequate synthesis). Assessment of the jugular venous pulse (JVP) provides a useful way of determining which of the two mechanisms is most important in a given case. A normal or low JVP suggests hypoalbuminemia as the cause, while an increased JVP points to raised hydrostatic pressure.

## Discussion of Case

The case of Mr. L.M. is a difficult diagnostic problem because of the unusual sequence of localized followed by generalized edema, the onset of both types of edema being subsequent to episodes involving gastrointestinal and constitutional symptoms of undetermined etiology. Because there was no evidence of lymphatic or venous obstruction to explain the penile edema with which he originally presented, the possibility of abnormal capillary permeability remained, although a specific cause could not be determined at the time.

When Mr. L.M. later presented with anasarca (gross generalized edema) he was found to be significantly hypoalbuminemic, leading to investigations aimed at assessing liver, gastrointestinal, and kidney function. Although the nephrotic syndrome is high on the list of potential causes of anasarca in a generally healthy middle-aged patient, it was ruled out by the absence of proteinuria. Protein-losing enteropathy arises as a result of excessive gastrointestinal (GI) protein loss and is associated with a myriad of disorders including lymphoma, inflammatory bowel disease, and tropical or non-tropical sprue. These considerations seemed attractive because of the associated GI symptoms preceeding each episode of edema. However, the fact that the nausea, vomiting, and diarrhea occurred during a discrete period of time and then disappeared while the edema persisted, despite a good appetite and a nutritional diet, makes this questionable. Additional investigations in the form of a normal upper GI series and abdominal ultrasound, negative stool cultures, and a normal alpha-one

antitrypsin level (a marker of intestinal protein loss) eliminated the possibility of a GI etiology. Although liver disease was entertained as a cause of the hypoalbuminemia, there was no real evidence to support this. There was no history of alcohol use or hepatitis, and liver function tests, enzymes, and ultrasound were all normal.

With the most likely etiologies eliminated, the category of increased hydrostatic pressure remained to be investigated. Primary salt and water retention due to renal insufficiency (decreased GFR due to chronic or acute renal failure) was ruled out by normal renal function tests and urinalysis. While the age and history alone made common causes of heart failure like coronary artery disease and chronic hypertension unlikely, rarer causes such as cardiomyopathies and pericardial disease were still possible. However, there were no symptoms of cardiac failure, the JVP was not increased, and the CXR was unremarkable, thus eliminating the heart as the source of the problem.

The only possible remaining mechanism, increased capillary permeability, was left as a diagnosis of exclusion. A deep skin biopsy showed supportive evidence of a diffuse angiitis, with a sparse perivenular infiltrate of lymphocytes and occasional giant cells. Although the underlying cause remains unknown, this process, generalized throughout the body, resulted in a leakiness of the vessels and the subsequent anasarca. The cause of the hypoalbuminemia therefore was not loss of protein in the urine or GI tract, but rather escape into the interstitium through the abnormal vessels. The patient was treated with oral prednisone, 60 mg/d, and showed a marked decrease in swelling and erythema within two weeks. The treatment is being continued until residual ankle and penile edema resolve, at which time the patient will be gradually weaned off the steroids. If a maintenance dose is required, low-dose steroids will be given q2d to prevent adverse effects of chronic therapy.

## Summary

While this patient has a very rare disorder, the mode of presentation is common to many different conditions. For this reason, it is important to have a rational approach to the work-up of a patient with edema, based on a knowledge of the possible etiologies and an understanding of the operative pathophysiological mechanisms in each.


## Acknowledgement

We wish to acknowledge Dr. A. Leznoff, whose insight and handling of this case led to the diagnosis.


## Further Reading


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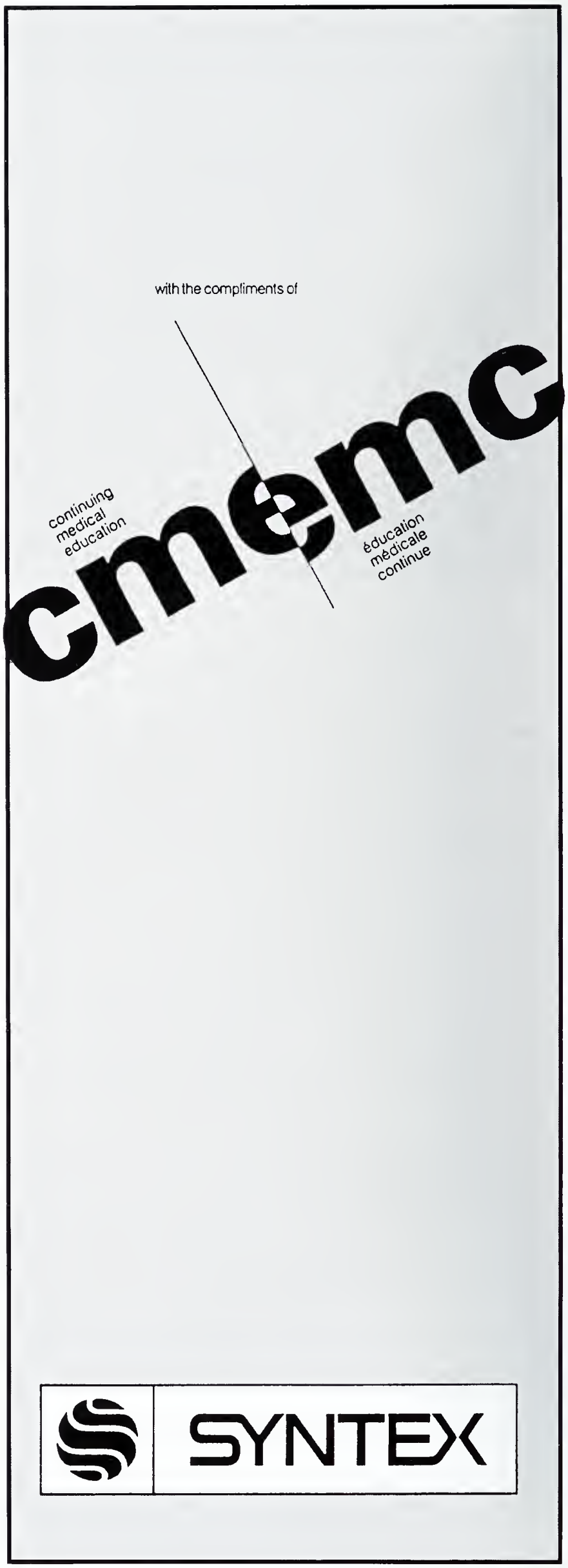


# Ophthalmology

*Andrew Taylor B.Sc. 9T1, in consultation with*
*Dr. G.E. Trope M.D. Ph.D. F.R.C.S.(C) F.R.C.S.(Edin.) Dip.Ophth.(Eng.)**
**Department of Ophthalmology, The Toronto Hospital, Toronto*

**Give a provisional diagnosis for each of the following cases:**

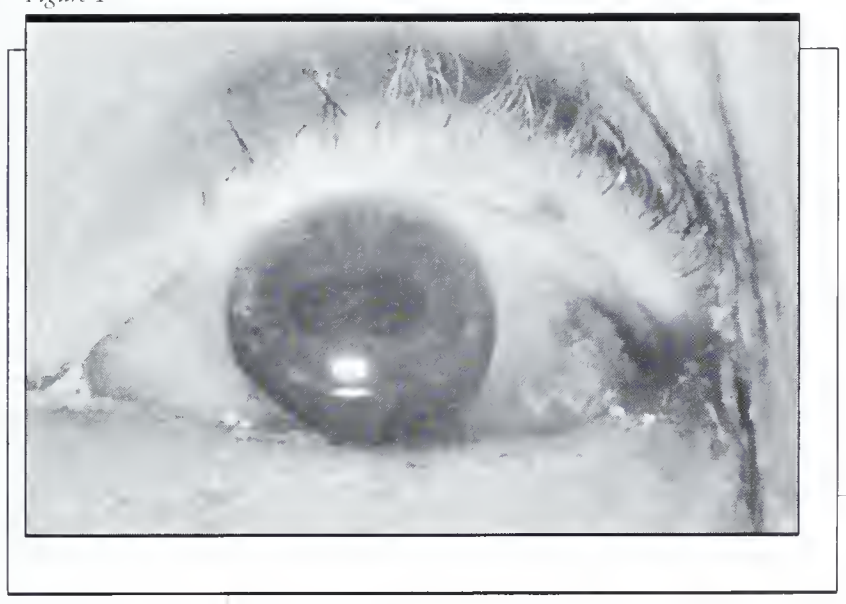
## Case 1

A 54 year old man presents with a painful left eye, decreased vision and photophobia of six days duration. On examination, the left eye appears as shown in Figure 1. Corrected visual acuity is: O.D. (oculus dexter or right eye) 6/6, O.S. (oculus sinister or left eye) 6/18. A haze in the anterior portion of the left globe impairs retinal ophthalmoscopy.

*Discussion*

The pupil is irregularly shaped and the surface details of the iris are obscured. The apparent haze must then be due to pathology anterior to the iris. Therefore, only two structures may be involved: the cornea and the aqueous fluid separating the cornea from the iris. The presence of the bright corneal reflex mitigates against a corneal etiology, suggesting pathology in the aqueous fluid of the anterior chamber. Together with the presence of a red eye, this indicates the presence of an inflammatory exudate consistent with anterior uveitis, or inflammation of the iris or ciliary body. The iris is inflamed, which causes both pupil and ciliary body spasm. The pupil is therefore small and the eye tender. Due to the presence of the inflammatory exudate, the pupillary margins can become adherent to the surface of the lens, a condition termed "posterior synechiae". This results in the peculiar shape of the pupil seen.

*Figure 1*



The history should elicit the presence of collagen-vascular disease, as well as possible exposure to toxoplasmosis, histoplasmosis, tuberculosis, and syphilis. In addition, any systemic nidus of infection should be looked for.

Treatment consists of dilating the pupil with atropine, local steroids to control inflammation, dark glasses for photophobia, and analgesics as needed.

*Key Words: red eye, visual loss, small sticky pupil.*

## Case 2

A 46 year old man presents with sudden blurring of vision and intense pain of the left eye, and vomiting. On examination, the left eye appears as shown in Figure 2. The right eye appears normal. Visual acuity is: O.D. 6/6, O.S. 6/60. The left cornea appears edematous with increased vascularity around its perimeter. Clouding anterior to the iris makes ophthalmoscopy impossible. A measure of intraocular pressure indicates: O.D. 12 mm Hg, O.S. 43 mm Hg.

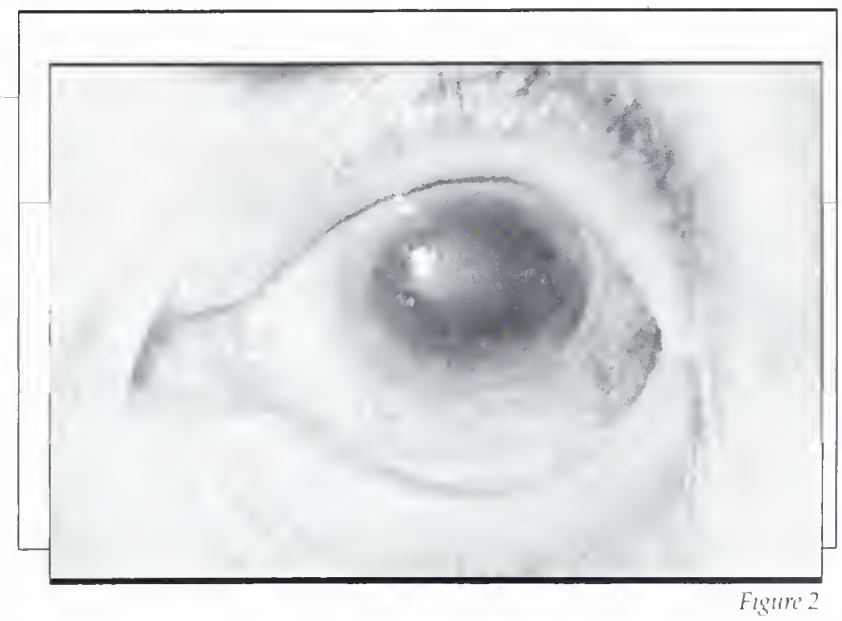


*Figure 2*

*Discussion*

Figure 2 shows a mid-dilated, oval left pupil and an edematous cornea with increased vascularity around its central perimeter. As in Case1, clouding anterior to the iris may be due to corneal or aqueous fluid pathology. In this instance, the corneal reflex is absent. Therefore, clouding must be due to pathology involving the cornea. An acute increase in intraocular pressure forces fluid into the cornea and causes corneal edema. This is pathognomonic of primary angle closure glaucoma (acute glaucoma).

Primary angle closure glaucoma is an ophthalmic surgical emergency. It necessitates immediate constriction of the pupil with a miotic agent, such as pilocarpine, to reestablish

aqueous outflow. In addition, an osmotic agent (e.g. glycerol), a beta blocker (e.g. Timoptic), and a carbonic acid anhydrase inhibitor (e.g. acetozolamide), to suppress aqueous fluid production, are administered. Definitive treatment consists of laser iridotomy once the attack has been broken.

*Key Words: mid-dilated vertically oval pupil, corneal edema, decreased visual acuity, pain.*

## Case 3

A 68 year old man presents with sudden loss of vision in his right eye. Flashing lights and black spots visual phenomena preceded the loss of vision by two days. On examination, there is complete loss of vision in the right eye. On ophthalmoscopy, the central portion of the right eye appears grey and the red reflex is absent. Figure 3 depicts the affected eye.

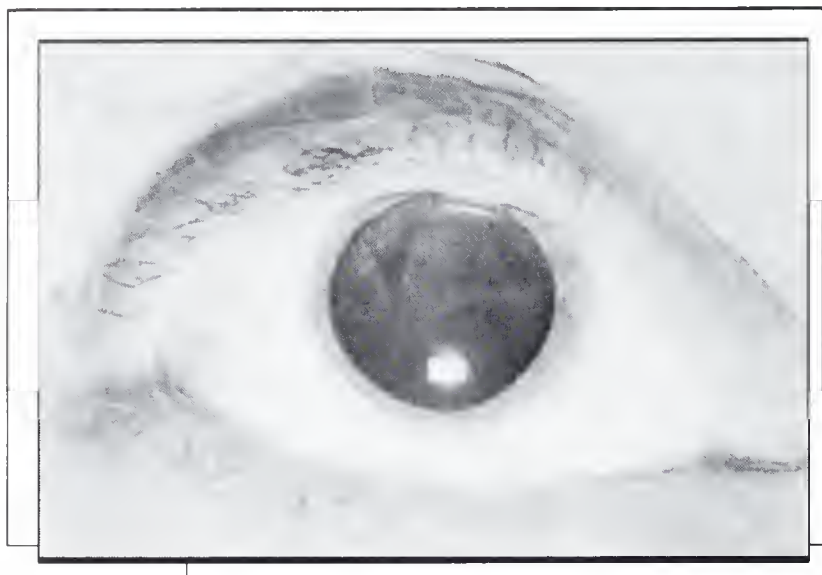


Figure 3

*Discussion*

Close examination of the eye shows a detached retina that can be seen in folds against the posterior surface of the lens. Loss of the red reflex and its replacement with a greyish colour should prompt one to consider a detached retina, especially if the loss of the red reflex is localized to one pole.

Retinal detachment begins with a localized retinal hole. The flashing lights seen by the patient are caused by vitreous traction on the retina as the hole is forming. Vitreous hemorrhages are created as the hole tears across small capillaries leading to the sensation of seeing black spots or cobwebs.

Figure 5

Immediate treatment of retinal detachment is required to prevent progression. In this case, the retinal detachment was complete at the time of presentation. Careful examination of the other eye must be made for retinal holes or vitreoretinal adhesions, the presence of which should be treated prophylactically.

## Case 4

An 18 year old female presents to emergency with a history of recurrent worsening of vision. Ten days earlier, she presented to her family doctor with sore red eyes. She was prescribed eyedrops containing a local steroid. On examination, the eye appears as shown in Fig. 4.

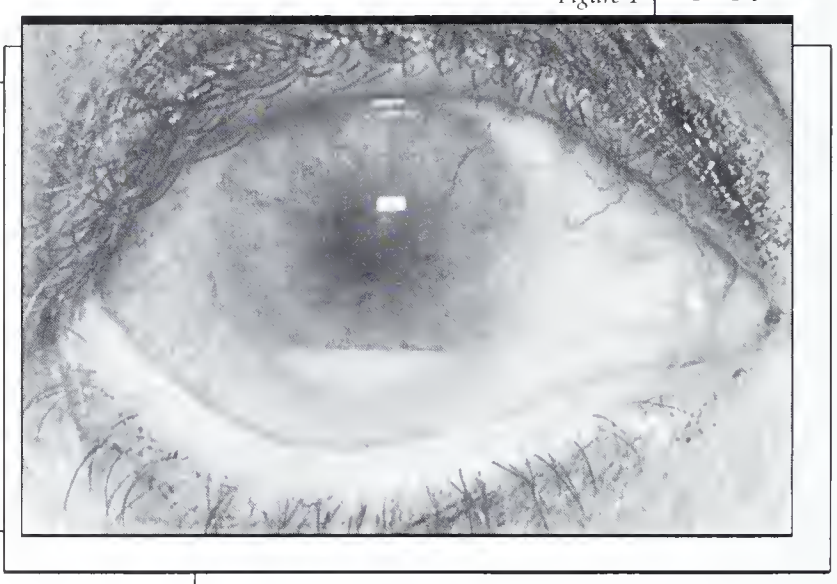


Figure 4

*Discussion*

Examination of her right eye demonstrates the presence of an hypopyon. The eye appears congested and a layer of white blood cells is evident at the base of the anterior chamber. An hypopyon results from a sterile or infectious intraocular inflammation (endophthalmitis) or can be seen as a complication of systemic collagen-vascular disease. Penetrating eye wounds of ophthalmic surgery can be complicated by infectious endophthalmitis. The presence of hypopyon necessitates urgent attention as the hypopyon ulcer can spread to the vitreous and result in pan-ophthalmitis. Topical and subconjunctival broad-spectrum antibiotics should be started immediately. This case demonstrates the danger in prescribing topical steroids before ruling out an infectious etiology of a red eye.

## Case 5

A 72 year old female complains of sudden loss of vision in her left eye. Corrected visual acuity is: O.D. 6/9, O.S. 6/300. The exteriors of both eyes appear normal. Ophthalmoscopy reveals absent red reflex in the inferior quadrant of the left eye, with replacement by a whitish colour. The fundoscopic picture is shown at left.

*Discussion*

The fundus demonstrates an occlusion of the lower branch of the central retinal artery. Note the pallor of the optic disc and the collapse of the artery in the inferior portion of the retina. The occlusion of the artery has led to ischemia and thickening of the retina in the affected area, preventing the choroid from showing through. Consequently, the retina appears white.

The etiology of this lesion is variable, but in an elderly patient one must consider temporal/giant cell arteritis. Other causes include: hypertension, arteriosclerosis, emboli and collagen vascular disease.

## Case 6

On routine ophthalmological examination, a 46 year old female, with no presenting complaints, demonstrates corrected visual acuity of: O.D. 6/6, O.S. 6/6. External examination of both eyes is unremarkable. The fundoscopic view of the right eye is shown in Figure 6.

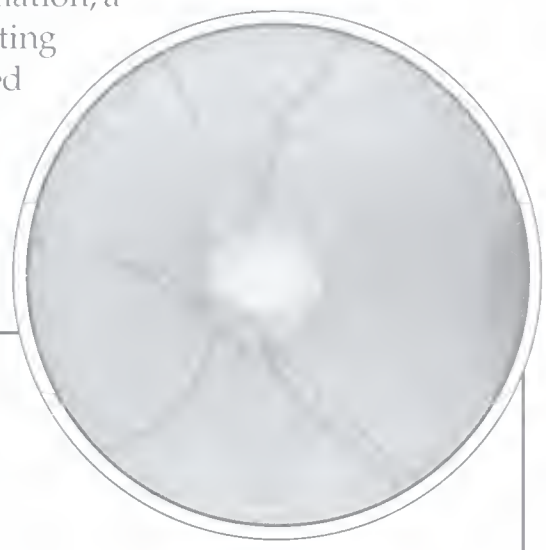


*Figure 6*

*Discussion*

This view of the fundus demonstrates a suspicious glaucomatous cup of primary open-angle glaucoma. The cup is enlarged with loss of the neuroretinal rim. It appears pale. There is some displacement of the disc vessels peripherally, and the vessels are seen to spill over the edge of the cup. The retina and the vessels appear normal.

Primary open-angle glaucoma is insidious in onset. Central visual loss occurs very late in the course of this disease. Thus, it is up to the primary care physician to detect the changes in the optic disc, the hallmark of this disease, for early diagnosis. Intraocular pressure is elevated as a result of obstruction of aqueous outflow due to an abnormality of the trabecular meshwork. If the pressure remains elevated, the optic nerve degenerates and the peripheral field vision is lost. Primary open-angle glaucoma is a bilateral disease, frequently presenting with one eye being more affected than the other. Family history is important as primary open-angle glaucoma is genetically determined.

Management entails follow-up readings of the intraocular pressure and visual field testing. Treatment consists of the use of eyedrops, e.g. miotic agents (primarily pilocarpine 1-4%) and beta blockers (Timoptic), laser trabeculoplasty or surgical trabeculectomy.

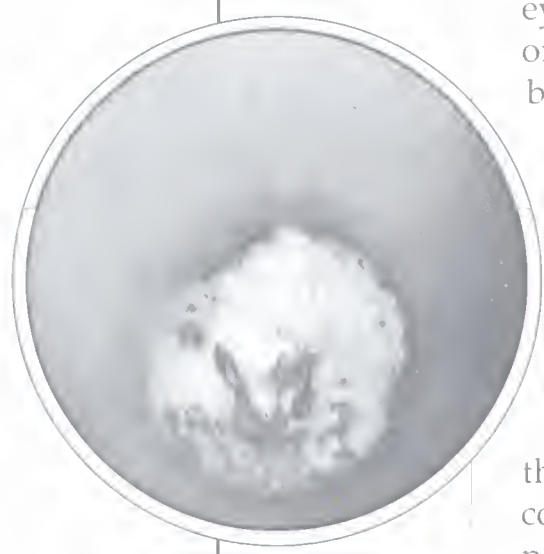


*Figure 7*

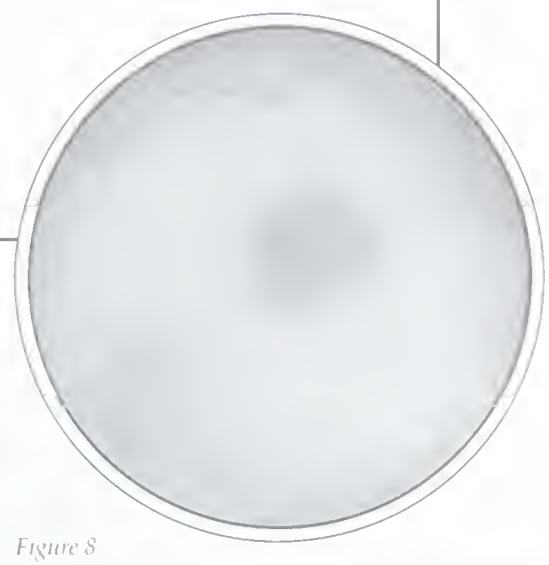


*Figure 8*

## Case 7

A 32 year old male presents with a 4-day history of decreased vision in his right eye. The fundoscopic appearance is depicted in Figure 7.

*Discussion*

This fundus is characteristic of chorioretinitis. Note the white scarring of the retina with proliferation of black pigment. This particular case is an example of Toxoplasma chorioretinitis. The presence of an active lesion often represents the reactivation of a congenital lesion. In 50% of Toxoplasma chorioretinitis, the macula is involved, causing a reduction of central vision in the affected eye. Other causes of chorioretinitis include: syphilis, tuberculosis, herpes simplex, histoplasmosis, candidiasis, toxocara and autoimmune diseases including Bechet's disease and sympathetic ophthalmia.

Treatment is directed towards the cause. When the macula or optic disc are threatened, the use of systemic corticosteroids may be employed. This will decrease the inflammation but not prevent scar formation.

## Case 8

Figure 8 depicts the fundus of a 16 year old female, on routine examination.

*Discussion*

This fundus demonstrates the presence of a flat, slate grey lesion underlying the retina. This lesion is characteristic of a benign choroidal nevus. A benign choroidal nevus may give rise to a malignant melanoma of the choroid, the most common primary intraocular malignant tumor. Continued observation is therefore essential.

# A Summer At The Baycrest Centre For Geriatric Care

*Eric M. Grief B.Sc. 9T1*

From discussions with students in my class, there seems to be the attitude that the elderly are a lost cause when it comes to helping them combat disease. When I told people that I had spent ten weeks working in a hospital where the average age of the patients is in the low 80's, the responses included: "Ugh", "Oh", and "What can you do for these people? They're too old!" In fact, very little of the curriculum at U of T deals with geriatrics. This lack in the curriculum leaves most students to their preconceived notions about the elderly and their place in society – this past summer, I had my eyes opened for me.

My goals this summer included becoming more comfortable being around patients, learning some general medicine, and exploring geriatrics as a career option. My responsibilities included attending morning rounds on the acute care ward, doing the admission history and physical, managing the patients that I admitted – with guidance from the interns, and compile data on those patients recovering on the ward. Otherwise, my time was free. This free time allowed me to read up on certain diseases that the patients had, visit clinics, and spend time exploring other parts of the Baycrest Centre. With second year clinical methods completed, I was eager to practice history and physicals. There were many opportunities for practice, and in the process I became much more comfortable talking with and examining patients in an unhurried and caring manner.

Morning rounds were educational, interesting, and sometimes exciting. Besides the staff internist, nurse, and myself there was a pharmacist, a physiotherapist, and two interns. Thus, a team approach was taken and this facilitated discussion of the patients' physical findings and subsequent plan of management. Questions relating to a patient's condition would often arise and I was frequently expected to answer, which kept me on my toes. Upon visiting with the patients, their progress and concerns were discussed. Often, the patients simply needed reassurance. Seminars were given regularly by the staff internist, the interns and geriatric residents, social worker, nutritionist, and communication disorder specialist. The topics covered in the seminars related closely to the patient population at the Baycrest and to the elderly in general and ranged from falls and confusional states to the importance of family conferencing.

When there were no new admissions, I spent the afternoons in clinics which are held in the day hospital. Being

*continued on page 32*

# Zambia: A Glimpse of the Dark Continent

*Lloyd Piszel 9T1*

I kept my eyes closed because the sun was shining so brightly. It was as if I hadn't seen daylight in weeks. As I walked from the infamous Drill Hall on St. George Street towards the post-exam celebrations, I was finally free to let my mind relax and wander to thoughts of the adventure that awaited me. For the very first time I realized that I was really going to Africa. After all the planning and organizing, and frustrations of putting together this trip, it was now only five days away!

The day of departure came quickly, a little too quickly as I still felt a bit tired from the grind of exams. After a tearful farewell at Pearson International, I was on my way; first to Amsterdam for a sixteen hour layover. I spent the day trying to find all the restaurants and bars I had visited the last time I was there, and after walking for ten hours, I figured I should give up. The next flight (another "all-nighter") brought me into the Dark Continent to Lilongwe, Malawi. As I went to the counter to check in for my final flight (to my destination Lusaka, Zambia), I suddenly remembered that I was on a waiting list. A wave of fear shook through me but was dispelled as I recalled the voice of my Toronto travel agent saying over and over, "Don't worry! Once they see you there, they will put you on. Trust me." While the airline clerk was checking the passenger list at the ticket counter, I kept my eyes closed, repeatedly mumbling, "Trust me. Trust me," out loud. I was rudely interrupted by those awful words that I will never forget, "I'm sorry sir, but we have no room for you on this flight, and all of our flights to Lusaka are booked up for the next month. There's nothing we can do for you." At that moment, my summer job had officially become an adventure, and I was a reluctant participant. Never being one to doubt my abilities in any situation, I decided that I could get out of this "minor" problem with a minimum of effort. Sometimes, I over-estimate my abilities, and this was one of those times. Still sleepless since leaving Toronto and being thrown into a mess of African bureaucracy, I was quick to learn that things are done differently here. The key words to remember are "slow" and "inefficient". To make a very long story short, I arrived in Lusaka after a two-day bus ride that made my second year immunology course seem like a treat.

So there I was in Lusaka, Zambia, the heart of South Central Africa; land of the Victoria Falls and Dr. Livingstone, the black rhino and home to over seventy tribal languages and cultures. I was there to experience it all. I had come to learn all I could about tropical medicine and

paediatrics, about Third World medicine, community health care, and AIDS and its devastating effects on this country. It was all so exciting, but it was too much, too soon! I hadn't slept more than a couple of hours in almost five days. I was starving and lonely and disoriented. I needed to get organized before I thought about any of those things I was going to accomplish. Indeed, the adjustment time was much more of a consideration than I thought it would be before I left. Finally, I tracked down my supervisor, the man I had been writing to for months, and got settled into my room. Now, I was ready to go.

The University Teaching Hospital in Lusaka is affiliated with the University of Zambia. The Department of Paediatrics is one of the largest departments on the hospital campus. It consists of an admitting ward, two general wards, an isolation ward, a malnutrition ward, along with a new neonatal surgical care centre (built by the Japanese in 1985). I spent my days on the wards in teaching clinics, seminars and rounds. The most common problems I saw were malaria, TB, sickle cell disease, rheumatic heart disease, pneumonia, a whole host of other tropical infections and of course, protein and energy malnutrition. The patient's symptoms and physical findings were always obvious, as parents would often wait to bring their children to the hospital until they were convinced it was the last resort. There would often be two or three children sharing one bed due to shortages, with their mothers sleeping on the floor beneath them. The wards were always crowded, noisy, and even chaotic, especially during visiting hours when hordes of relatives and friends would pour into the halls for their one hour visiting privilege per day.

With all the "happenings" in the hospital it was easy to forget the seriousness of these kids' diseases. There were just so many sick children all the time, that you had to be careful not to take too casual an approach to their illnesses. Although, on the other hand, I was constantly reminded of the preciousness of life as babies would die before my eyes. The death of a child in Zambia was as tragic as it would be in Toronto, and there was no difference between the strength of the bond of love between parents and child. Here, death was around me every day, but so was the strength of love and commitment in a family and the poignant struggle to survive.

While working here I encountered many problems that were at times difficult to endure. I expected things like diarrhea, terrible food and the occasional bout of homesickness, but it was the unexpected loneliness and total lack of anything to do after six o'clock in the evening that I found most difficult. This was all balanced by the warmth and friendliness extended to me by the other students and doctors, and the rather odd experiences I encountered by being regarded as a "curiosity" by many Zambians.

The education I received this summer was unique. Medically, I could not have asked for a better experience as I saw hundreds of patients with many diseases I will only read about in Canada. I was also able to "be a doctor" without the benefit of CT scans or echocardiography or many drugs we use routinely here. I had the opportunity to be a part of the celebrations and tragedies of life and to see how people half way around the world live and die. It was a summer filled with ups and downs, but given the chance I would do it all again.

A group of Zambian children outside the main hospital in Lusaka.

A typical scene at lunch time.

*Baycrest Centre, continued from page 30*

the only medical student in the hospital, I was free to attend any clinic and ask as many questions as I wished. The patients seen were from the hospital itself, from the adjacent Jewish Home For The Aged and affiliated Baycrest Terrace, or from the community at large. Among the clinics, Rheumatology, Dermatology, Neurology and Uro-Gynecology were excellent learning experiences since I saw patients on my own. The nurses and doctors were interested in my education and I witnessed procedures including cystoscopy, curettage of basal cell carcinomas, and debridement of wounds.

The patients in the clinics were very interesting in that they presented significant diagnostic challenges to the staff physicians. One 78 year old woman seen at the clinic presented with a ten year history of tremor, unstable gait, urinary incontinence, and declining ability to carry on a conversation with her husband. The differential diagnosis included Alzheimer's disease, normal pressure hydrocephalus, multi-infarct dementia, depression, and olivopontine cerebellar degeneration, among others. During the mental status exam she frequently broke down in tears exemplifying the frustration and fear that people experience as they realize what is happening to their ability to function normally. On physical examination she exhibited positive palmo-mental and grasp reflexes signifying frontal lobe involvement. There were no other definite findings. After observing this complete neurological assessment, I was able to assess a ward patient's mental status in detail. In summary, spending time in the clinics was useful. At most times patients were helpful and allowed me to examine them.

Activities outside the hospital were eye-opening. On a visit to the Baycrest Terrace, where the average age of the residents is 85, I was given a tour by an 80 year old woman. At the end of the tour I was invited back to this woman's apartment for tea and cookies. At the Terrace, the residents lead full and productive lives. While at the day clinic there, I met many vivacious and young-minded people who had lived through times of war and depression. Their spirit and love of life was great and made me realize that advanced chronological age is no reason to stop enjoying life.

While on the wards I learned a lot of general medicine. Every patient was an opportunity to learn. The classic symptoms of weakness and dizziness (or veak and dizzy – the vd's) were encountered often but since I was rarely rushed in eliciting a history, the vd's would usually turn into shortness of breath or pain on exertion. Moreover, I was taught to avoid the vitamin reflex where multivitamins are prescribed for the vd's because the elderly deserve the same full medical investigation as the younger population. Furthermore, one should avoid statements like "it's due to age" which takes away hope from the patient.

During the ten weeks, I reviewed much of second year cardiology in practical terms, namely, diagnosis and treatment. In the elderly, new onset shortness of breath can be the presenting symptom of a heart attack. When administering medications one is advised to 'start low and go slow'. By the end of ten weeks, I was beginning to wonder if all cardiac patients were in a state of "chronic Lasix (furosemide) deficiency".

Neurological disorders ran the gamut from TIA and stroke in evolution to Parkinsonism and dementia. In the elderly, delirium is often mislabelled as dementia. Since the elderly have little reserve, an acute illness such as a mild heart attack or just an acute change in environment can be enough to confuse them to the point of delirium. 'Sundowning' is when the elderly patient in new surroundings becomes confused as the sun goes down. The metabolic work-up for dementia was repeated often to rule out reversible causes of dementia. A word of caution – if the patient is having a hard time responding to your questions, make sure that their hearing aid is in place before ordering a full metabolic work-up.

Being on call was a very interesting way of knowing what lies ahead in clerkship. The first night on call will always be memorable for me because I answered a call while the intern was away, and later on in the morning after this 88 year old patient had stabilized, she began to vomit blood, drop her blood pressure, lose consciousness, and die in front of the eyes of the doctors, nurses, and myself. In fact, no heroic measures were taken and the patient was allowed to die with dignity. It was explained to me that people who go into cardiac arrest and who have underlying disease processes do very poorly – none survive for more than a little while. Having witnessed my first actual death, I spent some time reflecting on my own beliefs and read the book Jewish Reflections on Death. The second time on call, the intern was called to pronounce someone dead and I had the opportunity to just sit with the family member of the deceased. Being with a person who has just lost a relative is something that is not taught in school and I was fortunate to be able to help. All in all, the summer at the Baycrest was a thoroughly enjoyable learning experience.

## Acknowledgements

I would like to express my thanks to Dr. Michael Gordon and his parents for offering the summer scholarship. Also, my thanks to Dr. Marc Cheung, Sandy Wiesenthal and the entire 3-east team who helped me whenever they could.

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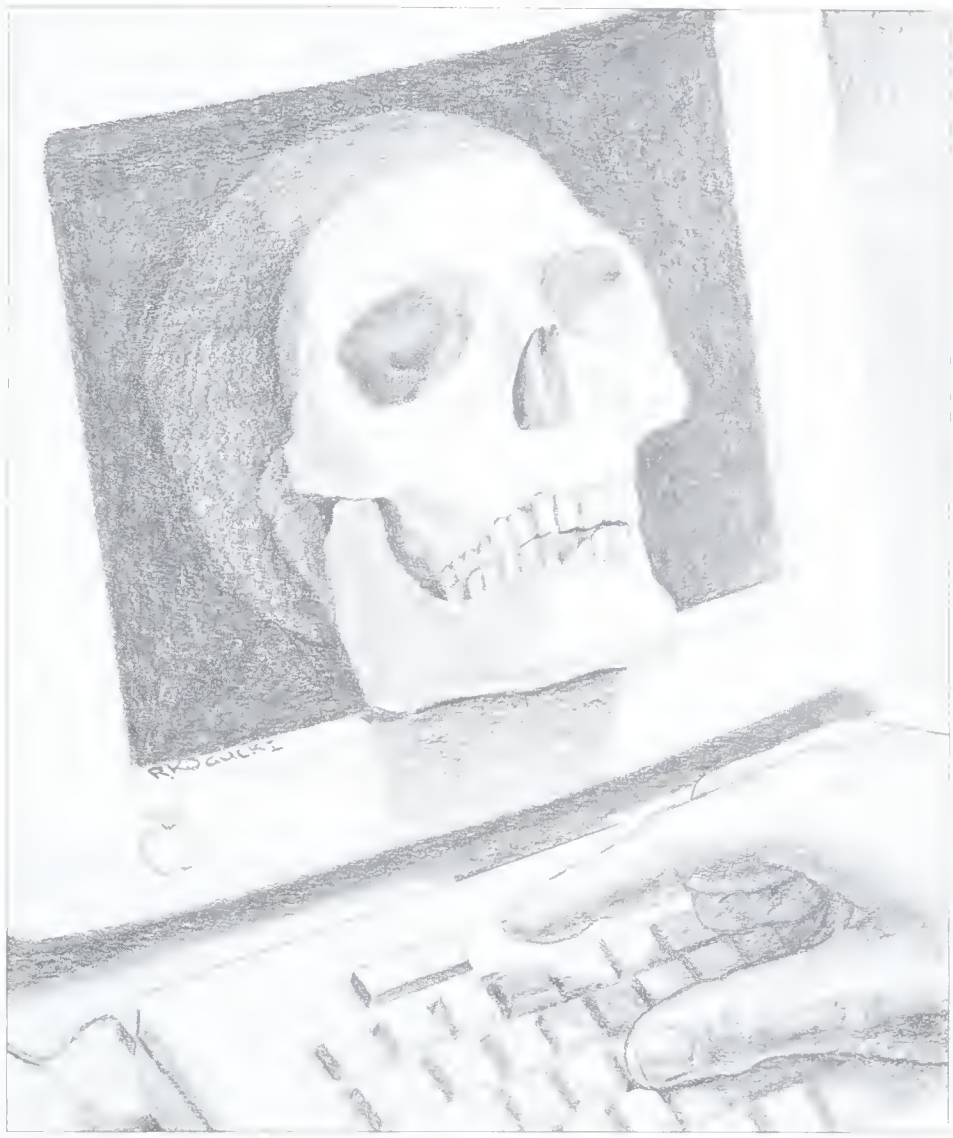
# The Psychiatric Interview: A Computer Assisted Instruction

*Mohammed Mahomed 9T2*



The task of conducting a psychiatric interview with a patient for the first time is a daunting challenge to many medical students. Even students who have conducted patient interviews on their own realize that practice is required to master the skills of psychiatric interviewing. Until recently, the only method of training medical students in the fine art of conducting an interview was by the means of role playing, with either students practising with hired professional actors or with each other. This made the availability of interviewing experience limited due to their cost and the limited time available in the curriculum for such training. With the development of laser disk-based Computer Assisted Instruction (C.A.I.) on how to conduct the initial psychiatric interview, students can now practise their interpersonal skills on their own initiative through an interactive format.

In the early days of C.A.I.-type software, information was presented in the form of text, with minimal use of graphics, accompanied by multiple choice questions. It was a means of teaching some basic concepts and skills while employing the novelty of personal computers to keep the users interested. As technology advanced, C.A.I. began to incorporate colour, graphics, and sound in order to make the presentation of information more enjoyable and interesting. Although this greatly increased the palatability of the concept, it still lacked the human interface which made such teaching seem artificial.

With the advent of laser disk ROM, the capacity of C.A.I. to teach was greatly expanded. Laser disk ROM is an optical disk player which can be connected to a personal computer by the means of a controller card. It enables the computer to use the optical disk as a massive database of read only memory, allowing the computer to recall immense amounts of data and pictures. The advantage of a laser disk ROM over other forms of read only mass storage devices is that it enables the computer to recall not only pictures, but entire sequences of film and video. This enables the animation of various computer applications with familiar vehicles such as people or cartoons. The use of this technology in C.A.I. has enabled their use in the teaching of various different subjects such as histology, pathology, immunology, and acute trauma care with realism and human contact.

The program under review here is the Psychiatric Interview: Module II in the Psychiatric Interview Series which was developed by the Louisiana State University Medical Center, Shreveport, Louisiana. The program requires an IBM PC with 640K, 20 MB hard drive, one floppy drive, IBM Info Window Colour Display Touch Screen Monitor and Controller, a Video Disk Player, and Dos 3.20 or higher. The program was developed by Lee Bairnsford, Ph.D. and Dennis A. Pernotto, Ph.D.

The software is an interactive system incorporating a simulated patient interview with easy to use touch screen menu bars and computer generated text screens. The program is divided into six main parts: INTRODUCTION, LISTEN, DIRECTING, RAPPORT, OBSERVE, and COMMENTARY. During each part, help is available by touching the on screen HELP bar on the bottom right corner of the screen. The help consists of four options: return to the main menu; accessing a glossary, which defines the various psychiatric terms used in the program; displaying an outline of the steps involved in a proper psychiatric interview; or resume from where the help function was accessed.

Each of the six main parts incorporates both video and computer functions to make it an interactive environment in which to learn. In the INTRODUCTION, the objectives of the course are clearly outlined and an explanation is given as to the operation of the software. The remaining five parts use portions of a simulated psychiatric interview in order to teach the student the proper method of conducting such an interview.

In the LISTEN module, the interview is presented to the student in stages. After each portion, the computer presents a multiple-choice question based on the information just presented in order to assess the listening skills of the user. If an incorrect choice is selected, the computer presents a reason for the inappropriateness of the choice and presents the section again, followed by the question, giving the user another chance.

The DIRECTING segment of the C.A.I. allows the student to choose which question or response the psychiatrist should use in each situation. Here, emphasis is placed on the student's ability to use open-ended questions, direct questions, and interpretation of patient responses correctly. Inappropriate choices are explained, just as in all other portions of the program, and another opportunity is given to pick the correct answer.

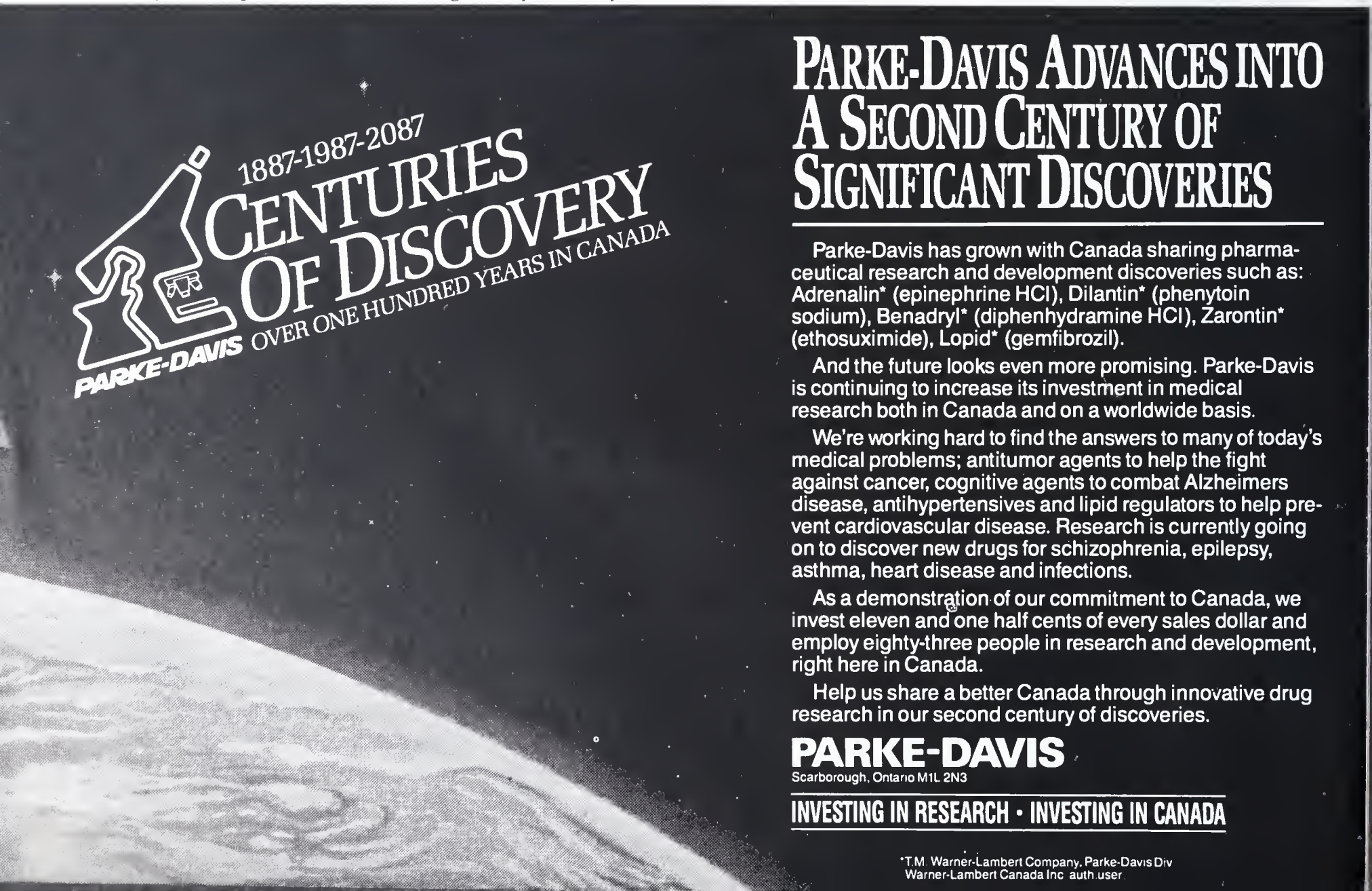
The RAPPORT portion of the program is similar to the directing portion. The student once again chooses the appropriate responses to the patient's comments, yet emphasizes the development of a solid doctor-patient relationship. This enables the student to learn how to respond warmly to the patient without being overly friendly and making the patient feel uncomfortable.

In the OBSERVING mode, the entire patient interview is presented without interruption. The purpose of this module is for the user to observe the non-verbal communication used between the patient and the physician and how it embellishes the process of interviewing patients.

In the final segment entitled COMMENTARY, the entire interview is presented once again, this time with the comments of the psychiatrist on crucial aspects of the interview superimposed on the sound track of the video. This provides a good summary of the interviewing process and highlights some of the key points in conducting successful patient interviews.

In essence, the program provides a good review of the general techniques necessary in performing patient interviews. With its interactive capabilities, it enables the student to gain some valuable practice in conducting interviews in a non-stressful environment. The software also provides feedback on inappropriate avenues of interviewing and enables one to discover the correct techniques by trial and error. Although the program becomes slightly tedious towards the end as the same case is used for each of the five main teaching segments, it provides one with the opportunity to practice the skills in this fine art so that one is more confident and adept at interviewing real patients in the hospital.

A copy of this C.A.I. is available to medical students through the Micro Computers Department at the Faculty of Medicine at the University of Toronto.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## Controversial Recommendations About Serum Cholesterol Testing

The role of elevated serum cholesterol and other lipid levels in the development of coronary artery disease (CAD) and peripheral vascular disease has been the subject of recent controversy in the popular press, and while most Canadian physicians and scientists who have studied the problem agree that elevated serum cholesterol is an important risk factor for heart disease, they cannot agree on the most effective measures to assess and reduce this risk factor in the Canadian public.

At the Canadian Consensus Conference on Cholesterol held in Ottawa in March 1988, physicians and scientists, expert in the field, evaluated the best available evidence and concluded that along with smoking, hypertension, diabetes and a family history of heart disease, serum cholesterol levels in excess of 5.2 mmol/L are a significant risk factor for coronary artery disease. These conclusions were based on the results of several studies, including the Multiple Risk Factor Intervention Trial (MRFIT), the Lipid Research Clinics Coronary Primary Prevention Trial and the Cholesterol Lowering Atherosclerosis Study. Furthermore, the results of these three studies, the Helsinki Heart Study and the National Heart, Lung and Blood Institute Type II Coronary Intervention Study have shown that reduction of blood cholesterol levels results in reduced rates of illness and death from CAD.

Based on these other studies, participants at the Canadian conference recommended several general and specific health measures for all Canadians. These included dietary guidelines, incentives to the agriculture and food industries to produce and offer foods that are low in fat and cholesterol, testing for serum cholesterol in persons at risk for other reasons and "...as resources permit, determination of lipid risk factors should become part of the periodic health examination for all adult Canadians".[1]

It is this last statement which appears to be the source of controversy. In 1988 the Scott Task Force on the Use and Provision of Medical Services in Ontario commissioned a Working Group on Cholesterol policy to evaluate the scientific data and to create guidelines on the management of cholesterol to be used by physicians in Ontario. The Working Group released these guidelines earlier this year, but they appear to be more conservative and less comprehensive than some experts in lipid disorders find acceptable.[2] The guidelines certainly do not recommend widespread screening of the public for serum cholesterol levels. Instead they urge physicians to assess cholesterol levels in patients with one or more other risk factors for CAD, including smoking, hypertension, diabetes mellitus, obesity, pre-existing CAD, and a dyslipidemia and/or myocardial infarction or CAD death in a first degree relative. Furthermore, the Working Group cut-off points for aggressive intervention and guidelines for the assessment and management of those in the intermediate-risk group for serum cholesterol are more conservative than some experts believe is appropriate.[3]

Critics of these guidelines have objected on scientific and other grounds that they are inadequate to be of widespread, long-term benefit.[3] On scientific grounds they argue that the recommended cut-off point for aggressive intervention is too low. Furthermore, they believe that greater efforts to determine other serum lipid levels in patients at intermediate risk for CAD based on their cholesterol levels are required than those found in the Working Group guidelines. Finally, there has also been criticism of the fact that there was limited access to the report before its release and that the group failed to consult the Ontario Medical Association's own expert panel on lipids before its approval.

For their part, the Working Group co-chairman Dr. David Naylor, an internist at the University of Toronto, and Dr. John Franck, an epidemiologist also at the University of Toronto, acknowledge the valid process-related criticisms of the report, but do not accept the scientific criticisms. They argue that the role of testing other lipoproteins in intermediate-risk patients is problematic because of inadequate standardization of testing in laboratories across the province. As a result, many persons may be wrongly classified for their risk and undergo unnecessary therapy.[4] Furthermore, Drs. Naylor and Franck argue that members of the Working Group may not have expertise in lipid disorders, but they are clinically and scientifically equipped to evaluate the available studies and to make recommendations based on their evaluation.

Critics of the guidelines, several of whom were participants at the Canadian Conference on Cholesterol, fear that because wider screening is certain to be costly, economic considerations have been given undue weight in formulating these guidelines at the expense of the future health of Canadians. In contrast, Working Group members believe that their guidelines represent a responsible, balanced scientific, medical and economic approach to the assessment and management of lipid disorders which, along with general recommendations to the public about smoking, diet and other risk factors for CAD, will significantly reduce morbidity and mortality in the future.

This important debate is likely to continue for some time to come.

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# Psychotherapy: Can It Delay Cancer Deaths?

For a number of years some scientists and clinicians have been intrigued by anecdotal evidence that a person's psychological or emotional state can significantly affect their response to illness and with it, their life expectancy. Consider for example, the familiar story of the bereaved spouse who, although otherwise well, dies soon after his or her partner. In the October 14th, 1989 issue of the British journal *Lancet*, Stanford University psychiatry professor, David Speigel, published a study which shows that psychotherapy not only reduced the anxiety and pain of a group of women with metastatic breast cancer, it extended their lives by an average of 18 months.

In the mid-1970's Spiegel began a study of the short-term effects of group psychotherapy on 86 women with metastatic breast cancer. The purpose of the study was not to get patients to "...wish away (their) illness...", according to Spiegel, but to help them face their mortality. Patients who received one year of psychotherapy were found to be less anxious and depressed than their counterparts who received only medical therapy. Recently Spiegel decided to follow up this group of patients, believing that he would find no difference in survival. When he gathered together the information on these women he was surprised to find that, while those in the control group lived an average of 19 months after joining the study, patients receiving psychotherapy lived an average of 37 months. Furthermore, the three women who were still alive more than 10 years after the study began were all in the group receiving psychotherapy.

In a recent news article in the American weekly journal *Science*, Boston University psychologist Bernard Fox, a strong critic of previous studies of the effect of the psychological state on cancer mortality, has expressed some reservations about the interpretation of the Spiegel study (see M. Baringa. 1989. Science 246:448). One important concern is that, in a study with only 86 patients, there may be hidden differences between the two groups which may explain the difference in survival.

Spiegel argues in response that while these are valid concerns, the two groups of patients were well matched for several important parameters, including ages at the time of diagnosis; time between diagnosis and appearance of metastases; time between metastases and entry into the study; and the kind and amount of surgery, chemotherapy and radiation the patients received.

Although sceptics like Fox have found Spiegel's study to be scientifically sound, a common concern of Spiegel, Fox and others is that these results will encourage some practitioners to forsake proven medical treatments in favour of psychotherapy. All the women in the study underwent standard medical and surgical treatment in addition to the psychotherapy given to half the group. Spiegel has pointed out that psychotherapy may not have had a direct effect on the patient's physiological responses to disease but may have changed their attitudes to medical therapy, resulting in better compliance with treatment regimes. It is this question which he hopes to address in future studies.

*Nick Pimlott Ph.D. 9T1*

## *Erratum*

The article published in Vol. LXVI, No. 2, pp. 18-22 entitled:

*How to Read a Chest X-Ray: A Systematic Approach,*

should have appeared as written by:
Michael K. McLennan, B.Sc. M.D.

instead of:
Michael K. McLennan, B.Sc. M.D. and
Michael J. Weinberg, M.Sc. 9T0


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# BOOK REVIEWS

## Physician's 1989 Drug Handbook

*1077 pp., Springhouse, Springhouse Corp., 1989; $29.95*

Both physicians and medical students need to know almost everything there is to know about drugs. From side effects, to dosage regimens, to pharmacodynamics and pharmacokinetics, this knowledge is absolutely essential in the medical field. Often, the only reliable source of such comprehensive information has been the Compendium of Pharmaceuticals and Specialties (CPS).

However, the inherent problem with the CPS is that it often requires one to spend valuable time searching through volumes of minutae to obtain the information one was interested in. In addition, the CPS is a very heavy and bulky book, and is therefore an unlikely candidate for a quick reference manual.

Well, now there is the Physician's 1989 Drug Handbook. This pocket-sized reference manual equals the clinical accuracy of a CPS but leaves its bulky, wordy nature behind. It not only provides information on every drug in clinical and experimental practice but does so in a very succinct and reliable fashion.

The drugs are listed alphabetically according to their generic name, underneath which appear their trade names. Finally, the pharmacologic classification of the drug (e.g. ß blocking agent), the therapeutic classification of the drug (e.g. anti-hypertensive) and the risk to pregnancy (if any) are presented.

The various headings range from indications, route and dosage to specific treatments for toxicities and overdoses. However, what makes this book stand out are a number of unique sections. One such section is entitled "Information for the Patient." In this section, there is a brief listing of information to give patients regarding the drug's uses and side effects.

Also very useful are sections outlining modifications required for paediatric and geriatric use and the adverse "Effects on Diagnostic Tests". However the selling point of this book is that it is so compact and concise that it can easily fit into one's lab jacket, hence the name Physicians 1989 Drug Handbook.

Physicians 1989 Drug Handbook does have a few drawbacks. For one, the book certainly won't win any prizes for the use of colour (all grey, white, and black) or graphics (there are few). Also, this book, although very good for quick reference, may not always provide medical students and physicians with all the answers they require and will necessitate the use of other references (most probably the CPS).

Nonetheless, there are no great drawbacks to this book and I would recommend it as a first-line reference text. It has helped me on numerous occasions when I did not wish to pull out my CPS (or carry it on the wards) and did not wish to read through volumes of minutae to obtain the information I required. And as this reference book is updated and revised every year it is a must for everyone.

*Mitesh Mehta 9T1*

## Cecil Essentials of Medicine

*Second Edition; Edited by Thomas E. Andreoli, Charles C.J. Carpenter, Fred Plum, and Lloyd H. Smith, Jr.; 830 pp., illustrated; Toronto, W.B. Saunders Company, 1990; $56.25*

Not surprisingly, in their search for answers, medical students have tried to find the ultimate textbook. This would provide the proper amount of detail crammed into a bound package that could be carried further than ten feet without experiencing shortness of breath (and all for a price that does not require choosing between paying for rent that month or for the book). Alas, one consequence of the rapid expansion of scientific and clinical knowledge has been the inability of a single textbook to meet the above requirements and, thus, displace Harrison's Principles of Internal Medicine (which certainly fails to meet the portability and affordability requirements) from its long-held position as "the textbook which no medical student should be without". The publisher of the encyclopaedic Cecil Textbook of Medicine (Harrison's chief competitor) has provided students with their idea of a textbook with the right amount of detail – a companion text entitled Cecil Essentials of Medicine, which according to its editors is a "succint and readable text that covers the indispensible elements of the principles and practice of the broad field of internal medicine." Upon receiving this book for review, I decided to use it while preparing for seminars on a variety of topics, rather than read the text from cover to cover. While this gave me a good chance to determine the usefulness of the textbook, it meant that some sections were examined in more detail than others. However, several students were also asked for their opinion of the book, and they consistently described it as the text they turn to first, but not exclusively.

The book is divided into 12 sections that focus on the major organ systems and diseases grouped under a common theme, such as infectious diseases or neoplastic disorders. Each section is further divided into chapters that describe the relevant signs and symptoms of each disease, important anatomic and physiologic considerations and the pertinent clinical and laboratory findings. Although there is nothing creative about this arrangement of information, it is logical and makes the textbook easy to use. Unfortunately, the index contains a less than adequate

number of listings, as some topics are discussed in the chapters but not referenced in the index.

The numerous and very clear tables, graphs and schematic diagrams are well described and referenced in the text and figure legends, although the lack of colours other than grey, burgandy, and pink, imparts a rather monotonous look to the whole book. In addition, some figures, particularly the ECG's and arteriograms are too small or poorly reproduced.

The editors have done a good job, as the core material is consistently well-written and no typographical errors were spotted. Cross references to other parts of the text are numerous, and the subject headings are large and easy to spot while skimming through the chapters. Each topic is adequately introduced and the information provided is, for the most part, up to date. Likewise, the pathophysiology and relevant clinical findings of the subjects are clearly and concisely presented and never force the readers to skim through paragraphs of trivial information before getting to the necessary facts. However, this companion textbook, while fairly good, is not likely to always provide medical students with the amount of information they desire, thus, forcing them to turn to the more detailed (and often frustrating) texts for a more complete story. Nonetheless, this book was never intended to be a complete reference text, but rather a succinct compilation of core material that is easy to read and understand. My impressions and those of other students are generally favourable, although Cecil Essentials of Medicine is more likely to please those students in the earlier undergraduate years (years one and two) who like to start with the basics and search out more detail only as needed.

*Elizabeth Quackenbush Ph.D. 9T1*

## Atlas of Microscopic Anatomy

*Written by R. Bergman, A. Affafi, and P. Heidger Jr.;*
*531 pp., illustrated;*
*Toronto, W.B. Saunders Company, 1989; $47.50*

The introductory study of Histology may be an unpleasant experience for the undergraduate medical student. It is possible that one day one might find a histological atlas that is so clear and concise and facilitates learning to such a degree that it actually makes Histology pleasant. But for now, we have to contend with such books as Atlas of Microscopic Anatomy.

Each chapter of this colour atlas begins with some introductory text which is then followed by a series of light micrographs (LM). There is usually one large light micrograph per page with some accompanying description underneath. The LM's are generally of quite good quality and are adequately labelled. The first five chapters discuss the basics such as cells and the various tissues (e.g., epithelial, connective, muscle, nervous, and blood). The rest of the chapters deal with organ systems such as cardiovascular, respiratory endocrine, and so forth.

An outstanding section in the atlas is the chapter on the central nervous system (CNS). The chapter contains a sequence of LM sections through the spinal cord, brainstem and cerebral hemispheres. This is excellent for introductory neuroscience courses and also serves as a good review before practical examinations in Neuroanatomy. There is also a useful glossary of terms used in describing the CNS at the end of the atlas.

Unfortunately, other parts of the atlas contain some deficiencies. One concern is the shortage of electron micrographs (EM). EM's are an integral part of modern Histology and their presence is necessary for better understanding. With the exception of the introduction, there are no EM's through the rest of the atlas. The authors excuse this deficiency by suggesting that one can find EM's in other supplementary Histology texts. However, this explanation is inconsistent because the authors have stated this book is intended to be a supplementary text as well as an introductory Histological atlas.

A subtle deficiency is the lack of figures or diagrams to facilitate better understanding of some of the basic principles of Histology. For example, the introductory text on Epithelial Tissues classifies the cell types and their subtypes adequately. However, this is immediately followed by a series of LM's from various epithelial tissues in the body, which to the beginner, may seem very confusing. However, the insertion of a diagram illustrating the various cell types immediately after the classification would help students construct a general abstraction, around which the LM's could be viewed and understood more adequately.

At times the authors mention various pathological conditions (for example pulmonary surfactant deficiency in neonates and chronic obstructive pulmonary disease) which are better left to histopathological atlases and not introductory ones such as this.

The authors of this atlas intended this book to stimulate learning in the field of Histology. It will, but not to a large degree. Overall, it is an adequate introductory atlas. However, there are better Histology atlases available. This one is better left on the shelf.

*Roger Sen 9T1*

## Books Received

The receipt of the following books is acknowledged. Books that appear to be of particular interest will be reviewed as space permits. However, this listing must be regarded as sufficient return for the sender.

1 *You Just Can't Hardly Believe It!* Wiley James. General Store Publishing House Inc., Burnstown, 1989.

2 *Canadian Medical Law: An Introduction for Physicians and other Health Care Professionals.* Sneiderman B, Irvine JC, Osborne PH. Carswell Publications, Toronto, 1989; $72.00.

3 *Nelson: Essentials of Paediatrics*. Behrman RE, Kliegman R. W.B. Saunders Companry, Toronto, 1990; $49.95.

4 *A Special Calling: My Life in Addiction Treatment and Care*. Bell GR. Stoddart Publications, Toronto, 1989.

5 *The Two Psychiatries: The Transformation of Psychiatric Work in Saskatchewan 1905-1984*. Dickinson HD. University of Regina, Regina, 1989, $32.00.

6 *Realize Your Dream*. Hines R. Deneau Publishers, Toronto, 1989; $35.95.

7 *Before We Are Born*. 3rd Edition. Moore K. W.B. Saunders Company, Toronto, 1989; $35.95.

8 *Essentials of Safe Dentistry for the Medically Comprised Patient*. McCarthy FM. W.B. Saunders Company, Toronto, 1989; $38.95.

9 *Joe Doupe: Bedside Physiologist*. Moore T. Ed: Morley, TP. Hannah Institute & Dundurn Press, Toronto, 1989; $17.95.


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**Abstract:** an abstract of 200 words maximum, typed on a separate page, is required for each article or note. No abbreviations should appear in the abstract. A translation of the abstract into French, if possible, should be included (abstract only).

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You CH, Lee K, Chey WY, Menguy R. Electogastrographic study: a patient with unexplained nausea, bloating and vomiting. Gastro-enterology 1980; 79:311-314.

For further details, see CMAJ 1985; 133:443-448.

**Tables:** should be numbered with arabic numerals, have a brief title, and be referred to within the text. Column headings and descriptive matters in tables should be brief. Vertical rules should not be used.

**Illustrations:** each figure should be planned to fit into the area of either one or two columns of text. Photographs and illustrations must be black and white and of good quality. Colour will only be considered if the author, or an outside sponsor, is willing to pay the cost. The figures (including halftones, photographs) are numbered consecutively in arabic numerals, and each one must be referred to in the text as well as being self-explanatory. Only essential labelling should be used with detailed information given in the caption. Letters and numerals should be of such size that the smallest character will not be less than 1 mm high when reduced.

**Drug Names:** both nonproprietry (generic) and trade names should be provided for all drugs mentioned.

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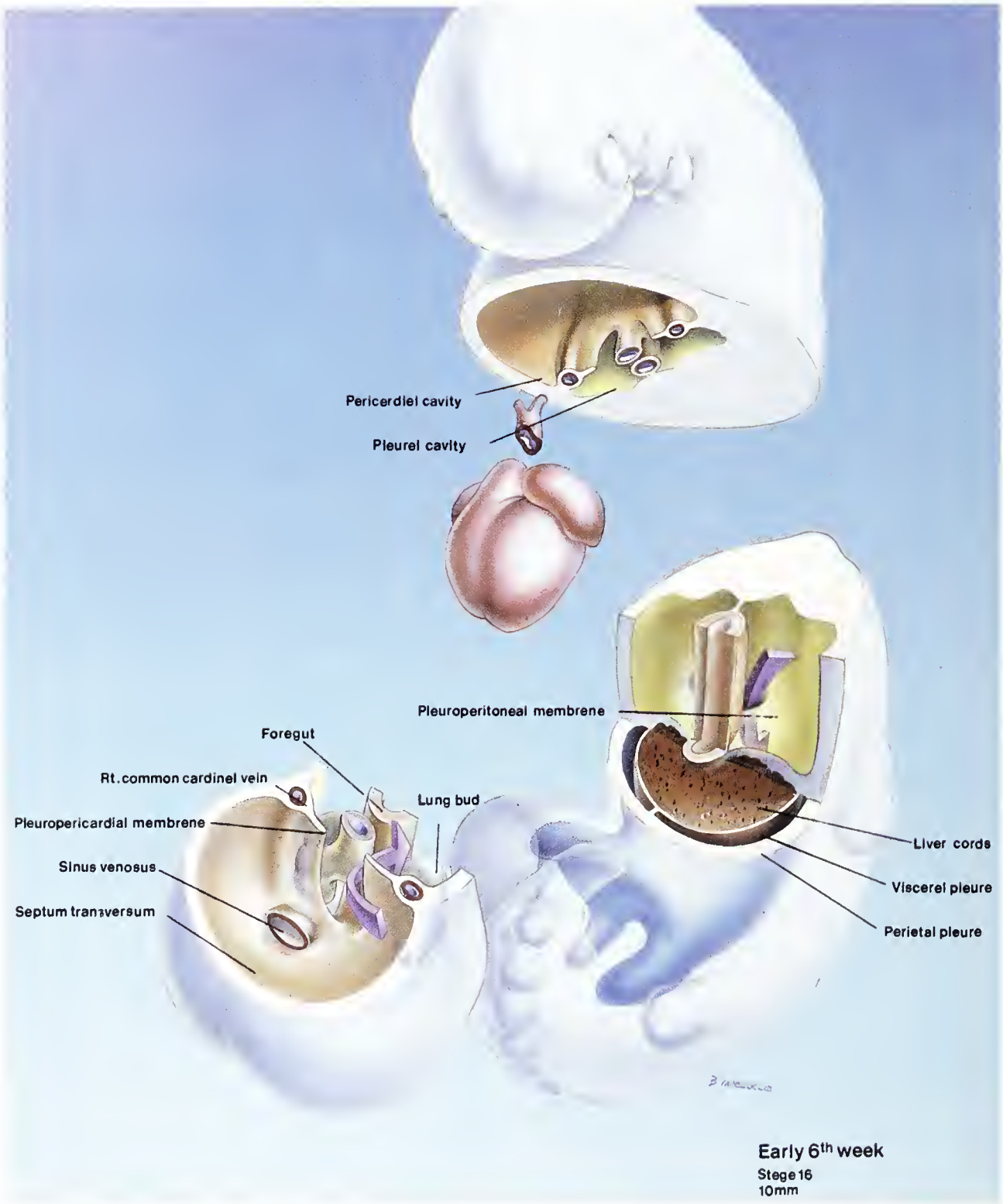


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